THE EFFECTS OF HEAT: THEIR TREATMENT AND PREVENTION WITH SPECIAL REFERENCE TO MILITARY PRACTICE.

by

F.M. RICHARDSON, M.B., Ch.B.

THESIS FOR THE DEGREE OF M.D.

THE EFFECTS OF HEAT: THEIR TREATMENT AND PREVENTION WITH SPECIAL REFERENCE TO MILITARY PRACTICE.

APPENDICES.

THE EFFECTS OF HEAT: THEIR TREATMENT AND PREVENTION WITH SPECIAL REFERENCE TO MILITARY PRACTICE.

The study of the Effects of Heat is one which has always engaged the attention of army medical officers as much of their service is usually spent in hot climates. A knowledge of the principles of temperature regulation has been given added importance in recent years by the practice of artificially inducing pyrexia for therapeutic purposes. It is therefore interesting to consider the abnormal conditions which result from failure of the heat-regulating mechanism, and to enquire what can be done to prevent or to mitigate their effects, and it is the purpose of this thesis to discuss this problem with special reference to military practice.

Recent Reports on the Health of the British Army show that "Effects of Heat" are responsible for a considerable amount of ill-health, and for some mortality, even when the British soldier in hot climates is leading the comparatively sheltered existence which is his lot in peacetime. The figures for India between 1930 and 1935 are as follows:-

EFFECTS/

EFFECTS OF HEAT IN INDIA. 1930 - 1935.

Year.	Heatstroke	Heat Examination — ? Heat Exhaustion	Deaths	Total Admissions
1930	26	102	13 (0.23)	128 (2.3)
1931	37	246	18 (0.32)	285 (5.1)
1932	50	178	18 (0.33)	229 (4.1)
1933	20	89	11 (0.2)	109 (2.0)
1934	11	77	9 (0.2)	90 (1.7)
1935	12	78	8	90

The figures in brackets represent the rates per thousand, which are not given in the report for 1935, but during that year more troops than usual had to remain in the plains during the hot weather, on account of military operations and civil disturbances.

From this indication of the incidence of these conditions in recent years, we can realise the enormous toll which heat has exacted from the British Army during the years in which it has lived and fought in hot climates. Any attempt to compare these statistics with those of former years would be valueless, and indeed misleading, for various reasons. Such a comparison would show an apparent increase in these conditions, - a conclusion which the improvements in the soldier's environment which have been made in recent years make it impossible to accept. The apparent/

apparent increase is undoubtedly the result of improved diagnosis, and of the inclusion of cases of Heat Exhaustion. In earlier years no exact figures were given for these conditions, and the cases were somewhat vaguely recorded under "Diseases of the Nervous System", or as "General Injuries", and the figures probably represent mainly the dramatic, and often rapidly fatal cases, which are nowadays in the minority. Many minor cases of Heat Exhaustion, which now help to swell the figures, would never, one feels convinced, have found their way to hospital in the sterner days of last century. One can imagine the surprise of the soldier of 1830, could he see a mild case of Heat Exhaustion being nursed in hospital, and, no doubt his medical adviser, too, might have something to say to his modern successor on the subject of "pampering".

Other cases, in which effects of heat may have developed in a patient under treatment in hospital, or where some of the varied prodromal symptoms of an insidiously developing Heat Stroke led to the clinician admitting and treating the case as one, for example, of gastric disturbance, were doubtless recorded under various other headings. The beneficial effect of the improvements in the soldier's environment is, however, reflected in the decline in mortality from

Effects/

Effects of Heat.

The figures for the year 1900 may be taken as an example of the high death rate from these conditions, at the beginning of the century, and similar figures are recorded for other years about that period.

1900	Cases	Deaths
Heat Stroke	95	74
Heat Apoplexy	67	32
Sunstroke	12	1

The death rate during the past few years compares very favourably with these figures which, it will be observed, show a great number of severe conditions, as compared with the present preponderance of Heat Exhaustion.

The incidence of these conditions may be expected to be higher in War than in Peace. In the South African War 1899 - 1902 the rate per thousand was 2.75 with 0.02 deaths, and during the Great War cases occurred in all the theatres of war. The vast amount of clerical work required, coupled with financial stringency following on the War prevented calculation of the rates per thousand, for all the forces engaged, but the incidence in some theatres of war was higher than in the South African War. In the Dardanelles the rate per thousand was 5.86 with no deaths; in South West Africa it was 12.30 with 0.03 deaths; whilst/

whilst in the period from 7th July to 28th July 1917 in Mesopotamia there were 3.445 cases with 482 deaths mostly amongst British troops.

If then, so many cases have occurred during the recent years of peace, and in campaigns during which rational clothing was worn, what can have been the incidence in less enlightened times? This question rises to one's mind, as, plodding through a snipe bog in Central India, sweating freely in a shirt and "shorts", one looks around at the hills crowned with forts once strongly fortified, and marvels at the endurance of the men who, under terrible climatic conditions reduced these forts, in months of fighting, wearing scarlet serge tunics with high leather stocks, and carrying heavy equipment, arms, and ammunition.

Of Sir Hugh Rose's campaign in Central India in 1858, the glories of which have always been, perhaps, somewhat overshadowed by the tragedies and heroisms of the first year of the Indian Mutiny, Malleson(1) says:-

"He and his gallant comrades had accomplished these great deeds during a season, the terrible heat of which far surpassed the heat of the corresponding season of previous years, and under a sun which proved scarcely less deadly than the enemy". A medical report by Dr Lowe says that "most of the officers and men were sick, and/

and the whole force needed rest..... other officers wounded or exhausted by the long and arduous duties and disease brought on by these and the terrible sun had been ordered to England", Dr Lowe is further quoted as follows "Officers and men fainted away or dropped down, as though struck by lightning, in the delirium of a sunstroke". Such, then, has been the lot of the British soldier at the hands of Heat and the study of how such disasters may be avoided must be the duty of every medical officer. Before considering the clinical conditions due to Heat, their treatment and prevention, the normal regulation of the body temperature must be briefly described, and the results of failure of this regulation, and the conditions which predispose to such failure must be considered.

REGULATION OF BODY TEMPERATURE.

The fact that "the temperature of a healthy man is fairly constant and does not differ more than a degree or two Fahrenheit, whether he be living in the Arctic Regions or in the Tropics", (Pembrey[2]) is sufficient proof that in the normal individual the heat regulating mechanism is extremely efficient. The normal range of temperature in the mouth is from 96.7° to 99°F. The rectal temperature which is from $\frac{1}{2}$ to $\frac{3}{4}^{\circ}$ higher is considered to be equivalent to the temperature of the blood in the Aorta[3], and is usually/

usually taken in cases suspected to be suffering from effects of heat. In hot climates the normal temperature may be from ½ to 1° higher than the figures given above, the evening temperature being especially liable to be on a higher level. The diurnal variation of 1° to 1.5°F, the evening temperature being higher than the morning, is due to the muscular activity of daily life, and is seen although in a less marked degree in a healthy man confined to bed, on account of the greater muscular activity in even rest in bed, than during sleep. That it is not due to the taking of food has been shown by the observation that the normal type of curve occurs in fasting men. Whan an individual takes to working by night, the temperature curve is gradually reversed, and is higher by night than by day. The regulation of body temperature is a faculty which man has gradually acquired, and in the infant it is less efficient, and the body temperature varies much more readily from a variety of causes. As the child grows, perfect heat regulation is acquired, and this fact together with the gradual alteration in the temperature curve of night workers indicates that training of the heat regulating mechanism, - or acclimatisation, is possible. Heat regulation is less perfect in the aged in whom an even temperature is found, and it may be said that absence of uniformity of/

of temperature is a sign of good health[2].

Even in well trained men severe exercise may cause considerable rise of temperature and after a three mile race, temperatures of 103°F to 105°F have been recorded in athletes.[4]. This rise of temperature is due to the heat liberated in the active muscles, for although part of the energy expended during active exercise is utilised by the Heart and Respiratory muscles etc., much of it is degraded into heat and lost by the skin and lungs. The loss of heat does not quite balance its production, and the temperature of the body therefore rises until a balance is reached at a higher level. This higher level of temperature is physiological, and helps to increase the rate of respiration and pulse rate in order to further the dissipation of the extra heat whilst it also assists the dissociation of oxyhaemoglobin in the tissues[5] with an accompanying increase of Carbon Dioxide and a rise in the hydrogen ion concentration. The beneficial effect of "warming up" is recognised by all athletes, and the optimum temperature is probably about 100.5°F, a temperature which helps to increase general metabolism, heightens the excitability of the medullary centres and possibly also facilitates the removal of lactic acid[6].

The temperature of the body, then, is the resultant/

resultant of heat generated and heat lost, and before further considering heat production and heat loss, the mechanism by which these two factors are balanced must be considered.

The association of disturbances of temperature regulation with lesions in various parts of the brain led to the assumption that there existed a cerebral centre or centres for the control of that function.

The actual site of the centre is according to Frazier, Alpers, and Lewy (7) in the substantia grisea in the floor of the Third Ventricle. They found this to be the site of the lesions in two cases of Tumours in the Pituitary area, in which death was accompanied by marked hyperthermia, and loss of temperature control, and in which the more lateral and dorsal parts of the hypothalamus were intact. Experiments on cats shewed complete loss of temperature control only when lesions were produced in the corresponding area, the nucleus hypothalamicus anterior. Minor disturbances, only, resulted from lesions in the lateral portions of the hypothalamus sparing the substantia grisea, and unilateral lesions in this vital area produced severe, but not permanent disturbances of temperature control. In experiments with the Rhesus monkey, Macaca Mulatta, Ranson and Ingram (8) found that damage to the Thalamus alone did not upset the regulation of temperature, whilst/

whilst bilateral destruction of the caudal part of the lateral hypothalamus caused prolonged incapacity to keep the body temperature up to normal. These and other observations indicate that the vital area in the Brain, for temperature control is in the hypothalamus, and the view previously held that it was in the Corpus Striatum has been further disproved by the observation that removal of the Corpus Striatum was not followed by loss of temperature control.(4) (9)
In the experiments of Barbour, therefore, in which the Corpus Striatum was heated or cooled by tubes of hot or cold water,(10) the fact that responses in the heat regulating mechanism were only produced by degrees of temperature considerably above or below that of the body, was due to the necessity of affecting the vital centre some little distance away. Further, the temperature disturbances recorded in association with lesions of various other parts of the Brain can probably be attributed to pressure effects on this vital area, or to interference with its blood supply. It is uncertain whether the centre most commonly receives its information about alterations in body temperature from afferent nervous impulses arising in the temperature sense organs, or from alterations in blood temperature. In the case of reactions to cold, it would appear that nervous impulses may convey the information/

information to the centre, for the rapidity with which cooling of the sensitive snout of a dog leads to cutaneous vasoconstriction suggests a more rapid action than that of cooled blood affecting the centre.[11] So rapid a response to a rise in body temperature, is however, not often required, for we have already seen that a moderate rise in temperature may be actually beneficial, assisting as it does in the efficient performance of muscular work. The importance of alterations in blood temperature is suggested by the observation that in an individual with complete destruction of the spinal cord in the mid dorsal region, vaso dilatation or vaso constriction in the face and hands occur in response to immersion of the paralysed limbs in hot or cold water just as in the normal subject[12]. In the case of extensive paralysis, as in pontine haemorrhage, the paralysed parts, cut off from the heat regulating centre, passively follow the temperature of their environment.

The appropriate responses, constriction or dilatation of the cutaneous arterioles, and the initiation of sweat gland activity, are produced by inhibitory or excitory impulses transmitted by the sympathetic nervous system. That these impulses originate primarily from the so called temperature regulating centre, is, however, disputed by W. Cramer[13], who maintains/

maintains that the centre is merely "a group of nerve cells representing the central connections of the sympathetic", and holds that it is the Thyroid Adrenal apparatus acting through the sympathetic nervous system which is of major importance in temperature regulation. The existence of this "heat regulating mechanism ancillary to the nervous apparatus" is, he points out, indicated by the observation that "even the most completely destructive lesion", in the hypothalamus, "does not render an animal, such as the rabbit, completely poikilothermic. If the thermal environment is kept at 28°C such an animal can still maintain its normal body temperature at about 38°C".

On exposure to cold the Thyroid and Adrenal glands are stimulated, and through the sympathetic nervous system increase metabolism, thus increasing heat production, whilst they diminish heat loss by constriction of the cutaneous arterioles. Change from a hot to a cold environment is accompanied by diminished cellular activity of these glands. The suddenness of the change in the thermal environment, no less than the actual degree of the temperature acts as a stimulus to the glands.

It has long been recognised that the Thyroid Gland has an influence on the regulation of body temperature, a fact to which the disturbances of temperature in Myxoedema early drew attention, and which/

which recent experiments have served to confirm. Thus the Thyroid gland has been shown to respond immediately to changes in temperature by alterations in its rate of oxygen consumption, and in the rate of blood flow through it. If the body, or the carotid blood, is warmed the rates of blood flow and of oxygen consumption in the Thyroid fall, whilst cooling causes them to rise. Similar responses to the fever induced by Tetrahydro β naphthylamene occur. Administration of thyroxine alters the responses obtained in experimental animals to drugs which influence their body temperature. Fever-producing drugs in rabbits after thyroxine administration cause fatal hyperpyrexia whilst the fall in temperature resulting from luminal is reduced or abolished.(14) Histological changes indicating functional activity occur in the Thyroid in experimental animals in a cold environment, and on exposure to a temperature of 37°C the gland passes into a resting state similar to that which occurs after Thyroid feeding. It seems probable therefore that external cold does stimulate Thyroid secretion, with a consequent increase in metabolism, and that a rise of body temperature results in a reduction of the secretory activity of the Thyroid and Suprarenal medulla, by which means the metabolic activity of the body is reduced, or its tendency to rise with body temperature/

temperature is compensated. Whether this effect on the Thyroid and suprarenal is produced by nervous influences or by alterations in the blood temperature is uncertain. Although proof of the relation of the Thyroid with the suprarenal glands is less firmly established than of its relationship with the Pituitary it seems probable that in connection with heat regulation, the glands are closely related. The calorigenic action of adrenaline, which in response to external cold decreases heat loss and stimulates tissue metabolism is diminished after Thyroidectomy. This, and the resemblance between the effects of hyperthyroidism and those of sympathetic stimulation, suggest that the Thyroid's contribution to temperature regulation may be to increase the secretion of adrenaline, or to sensitize the mechanism on which adrenaline acts. That the suprarenal cortex may exercise an inhibitory control over Thyroid activity is suggested by the observation that the increase of heat production observed after injury to the suprarenal cortex does not occur in thyroidectomised animals,[15] and also that the administration of Thyroid has been found to cause hypertrophy of the suprarenals. Haemorrhage into the suprarenals has long been known to be associated with a high temperature. Thomson[16] described the condition as frequent/

frequent in the new born as a result of birth injury, and occurring also in children a few days old as a result of septicaemia. The most interesting type of case he described as occurring in the first year of life as part of a rapidly fatal illness characterised by high fever, severe abdominal pain and sometimes nausea and vomiting. Such cases are referred to by Thursfield and Paterson[17] and by Lakin[18] who has "met with the condition in the post mortem room, not only in infants, but once or twice in adults with Pneumonia", and who also points out that in certain rare cases of pyrexia of unknown origin, adrenal haemorrhage has been the only lesion found in post mortem. Crile[19] described acute adrenal deficiency as being characterised amongst other things by "a sustained high temperature in the absence of infection". There is therefore much to support the contention of Cramer that the activity of the Thyroid Adrenal apparatus is of importance in temperature regulation, but the gulf between his theory and that of a purely nervous control is not a wide one and a means of bridging it is suggested by the views of Lee[20]. Lee describes as "Primary active adaptations" to hot environments, the dilatation of cutaneous arterioles, initiation of sweat gland activity, and increase of blood volume, resulting from stimulation of the heat regulating/

regulating centre. If these measures are insufficient to maintain heat loss the body temperature rises, the excretory activity of the Thyroid and Suprarenal medulla is reduced in response either to nervous influences or to alterations in the blood temperature, and thus the metabolic activity of the body, as we have already seen is lowered, or its rise with body temperature prevented. Lee's view that this alteration in endocrine balance is probably one of the chief factors in acclimatisation is in agreement with the view of Cramer who states that "the intrinsic mechanism of heat regulation is a function of the sympathetic nervous system. The secretion of the Thyroid and Adrenal hormones reinforces and prolongs the activity of the Nervous System".

Such a harmonising of the two theories of temperature regulation is attractive, and, it may be added, that the fact that there is a long latent period before Thyroid secretion produces its effect, suggests that that gland, at least, is more adapted to aiding acclimatisation than to acting as a safety valve for response to any sudden need of the body for temperature regulation.

HEAT PRODUCTION.

Heat is generated by all the oxidative processes taking place in the body, but mainly in the skeletal muscles and, to a lesser extent, in glands, especially in the Liver and Kidneys.

The rate of oxidation in the tissues is so adjusted as to maintain the body temperature within normal limits. Wright[4] points out that the basal metabolism is closely related to surface area, and estimates that 40 calories are given off every hour per square metre of body surface, the surface area of an average adult being about 1.8 square metres. He quotes De Almeida as saying that in the Tropics heat production is only about 30 calories per square metre as compared with 40 in temperate climates, and Martin[21] who also noted this difference found that the adjustment was a gradual one and varied somewhat with individuals. Heat production is less in old age than in youth, apart from differences in muscular activity. The intake of any of the three constituents of food is accompanied by a rise in the rate of the resting metabolism, and the increased combustion due to proteins is greater and lasts longer. There is considerable variation in the rate of oxygen consumption of different tissues, resting muscle using least and Kidney most. Thus the heat production of resting muscle/

per what unit?

muscle has been found to be about one large Caloric per hour, that of Liver, Pancreas and Submaxillary gland about 8 and of Kidney about 25 Kilo calories[21]. Pembrey[2] estimates that the minimum heat production of an 11 stone adult male is about 1700 Calories in 24 hours, of which 500 calories are due to glandular and 1200 to muscular activity. The unceasing activity of the Liver and Kidneys, according to Tigerstedt quoted by Pembrey, is responsible for 368 and 74 Calories respectively; the respiratory muscles produce about 150 Calories, the Heart about 70 Calories, and the balance of about 1000 Calories must be produced almost entirely by the skeletal muscles. The generation of heat in organs and glands is not under voluntary control, except as regards the nature, temperature and amount of food which is taken. The Liver is the most important of these cources of heat, and to it Cramer[13] ascribes the chief importance in chemical heat regulation - that is in the processes concerned with variations in heat production, as opposed to physical heat regulation, which is concerned with variations in heat loss. The glycogenic function of the Liver is responsible, under sympathetic control, for the provision of the fuel needed for increased heat production. The heat produced by the constant metabolic activity of the Liver itself, is, as we have seen, considerable, and from the point of view of/

of heat distribution the Liver is well situated, receiving, in addition to its own blood supply, the blood from the Portal system on its way to the Heart. The Kidneys also are in a state of constant activity, and despite their large blood supply, the blood leaving them is $^{1}/_{10}$th of a degree Centigrade hotter than that entering them[11]. As regards the 70 Calories ascribed to the Heart's action it was found by Snyder[22] that heat production in Auricular and Ventricular muscle shews considerable variation at times, and in successive beats, and is generally greater in oxygen plenty than in oxygen want.

It has already been stated that the chief source of heat is the skeletal muscles, and heat is generated in these not only during voluntary exercise but also to some extent during deep sleep or anaesthesia by the maintenance of tone. The more or less involuntary muscular action of shivering is directed to the rapid production of heat, and increased heat production has been observed in association with the spastic state resulting from extirpation of the motor and premotor areas in monkeys[23]. In the case of a lower motor neurone paralysis, whilst there is at first a rise of temperature in the paralysed limb, due to loss of vasomotor control allowing more blood to circulate through the dilated vessels, and to cessation of sweating/

sweating, the paralysed limb soon becomes colder than the healthy one, since heat production ceases in the flaccid muscles and the flow of blood is lessened by the local vasomotor tone which asserts itself in a few days and by the impaired venous return due to the absence of muscular contraction[2].

The heat evolved is due to a number of complex chemical changes occurring during contraction which include the breaking down of glycogen, neutralisation of the resultant lactic acid, and the breaking down of phosphagen. Resynthesis of phosphagen is however, an endothermic reaction, and requires a supply of energy. The evolution of heat occurs during contraction and relaxation, and also, especially when the muscle is stimulated in the presence of oxygen, after the contraction is over. The rise in body temperature, which has been referred to, is less in the trained than in the untrained man, on account of the increase in mechanical efficiency which can be effected by training. We have seen that much of the energy liberated in muscular work is dissipated as waste heat, and the ratio of the energy converted into work to the total energy liberated indicates the mechanical efficiency of the subject. Various estimations of the degree of mechanical efficiency have been made by different investigators. Whilst Martin[21] finds that/

that the mechanical efficiency of the most practiced muscular action is only about 20%, Wright[4] finds that in athletes the mechanical efficiency is 25 to 30% compared with 20 to 23% ~~compared with 20 to 23%~~ in the untrained man.

When severe exercise is completed the rate of oxygen consumption remains for some time higher than the normal resting level. The amount of this excess of oxygen utilisation over the normal figures for the same length of time is known as the oxygen debt, and represents the excess of oxygen required for a piece of work over the oxygen used during that work. During severe exercise therefore, not only does the body temperature rise during the exercise, as heat loss cannot keep pace with heat production, but, on account of the accumulated oxygen debt, heat continues to be evolved after the cessation of the exercise, and a continued rise of body temperature may occur.

As we have seen, muscular exercise may cause the body temperature to rise to as much as 103° to 105°F in the normal subject, and Martin's[21] calculations give an indication of the amount of heat which may have to be dissipated. Walking at four miles per hour, he found, requires about as much oxygen consumption as performing work on an ergometer at $^{1}/_{10}$ horse power, and so causes the same amount of heat output per minute. This amount, allowing for a mechanical/

mechanical efficiency of only 20% is 4.35 large calories which added to an average resting heat makes about $5\frac{1}{2}$ large calories per what? . If the work is performed in the sun another 4 or 5 calories may have to be added. In a climate, therefore, the shade temperature of which is at or above that of the skin, 10 large calories per minute have to be removed from the body. Numerous experiments on oxygen consumption at different temperatures have shown that the rate of metabolism is increased by exposure to cold even before shivering occurs, and as already stated there is a tendency for basal metabolism to be lowered by hot climates. Such variations of heat production or chemical heat regulation, in which the Thyroid Adrenal apparatus may play a leading part, is however of most value in the response to a cold environment. Protection from the adverse effects of a hot environment needs more than mere reduction of heat production, especially in persons who must lead an active life and the loss of heat by the body, or physical heat regulation, must now be considered.

HEAT/

HEAT LOSS.

It was seen when discussing heat production and mechanical efficiency that much of the heat produced by metabolic processes, and by muscular action is dissipated as waste heat. At ordinary comfortable environmental temperatures and with activity short of physical labour this heat is got rid of by radiation, conduction, and convection from the body surface, by evaporation from the Lungs and Skin, by warming of the inspired air and of food, and by the warm excreta leaving the body. The parts played by the different factors have been variously assessed but it may be said that radiation convection and conduction account for about 75% of the heat loss, and the evaporation of water from the Skin and Lungs for about 20 to 25%, the losses by the other routes being comparatively trivial (2) (4) (11) (21). In hot weather or with heat production increased by hard work the amount lost by evaporation of water rapidly increases, and may be responsible for as much as 100% of the total loss. Evaporation therefore is most important in hot climates, whilst in cold or cool climates radiation and convection are important. Since liquids conduct better than gases the importance of conduction is greater in a moist than in a dry atmosphere. The influence of atmospheric conditions on heat loss will later be described/

described in greater detail, and now the physiological response to a need for increased heat loss must be discussed. For the equalization of temperature in living organisms a mobile constituent which can circulate freely and rapidly is most desirable, and water, which because of its high specific heat can absorb large amounts of heat, is the fluid best suited to this purpose[24]. Because of its fluid nature water can distribute equally throughout the body the heat which it has absorbed from the source of its production, and can carry any excess to the surface, there to be given off. Water therefore acts as a buffer in high temperature changes, and as a vehicle of heat within the body, and the equalization of temperature is attained by water shifting under nervous control.

The first reactions to a hot environment, called by Lee[20] "immediate passive reactions", are a rise in skin temperature, and a resultant increase in evaporation of surface moisture, or as it is often called "insensible perspiration". These reactions are independent of life, but now, as a result of stimulation of the temperature-regulating centre, active adaptations occur. Cutaneous vaso-dilatation exposes a sheet of blood to the cooling effects of the air, the increased vascularity of the skin promoting loss/

loss of heat not only by radiation, convection, and conduction, but also by the evaporation of surface moisture. If this is not sufficient to control the rise of temperature the sweat glands are stimulated to activity and visible beads of sweat are produced. The Pulmonary ventilation may be somewhat increased, and the secretory activity of the Thyroid and Suprarenal medulla reduced. The activity of the individual is lessened, his inclination for food and work decreases, and the basal metabolic rate may be lowered. To produce the increase of blood-flow through the skin, which Grollmann[25] found to be approximately 700 cc per minute at an external temperature of 45°C, the volume 113°F of the abdominal organs and the Brain is diminished[26], and there is a considerable rise in the rate and minute output of the Heart. The Heart rate may show an increase before any rise of body temperature occurs, probably from nervous influences, and once the body temperature rises the rate increases rapidly[20]. Grollmann[25] found that the effect of increasing temperature on the pulse rate is progressively greater at elevated temperatures - thus there was an increase of 6 beats per minute from 20° to 30°C, of 9 beats from 30° to 40°C and of 11 beats from 40° to 45°C. The increase follows changes in cardiac output fairly closely so that the stroke volume is fairly constant. The/

The systolic blood pressure is much less variable than the cardiac output and pulse rate, declining from 116 at $0^{o}C$ to 100 at $32^{o}C$ where it remains, and rises again slightly at $45^{o}C$. The pulse pressure is fairly constant. Incidentally Grollmann's observations led him to believe that the blood pressure is in general much less variable than is generally assumed, and as his observations were made under basal conditions he concludes that if deductions from blood pressure readings are to be of value more attention should be paid to this factor. That the increased output is directed to procure heat loss and not for metabolic activity is indicated by the high oxygen saturation of the venous blood leaving the skin. The increased venous return necessary to maintain the raised minute output is facilitated by the raised capillary and venous pressure resulting from the arteriolar dilatation in the skin. The relative importance of the extremities in this cutaneous vaso dilatation is shown by the observations of Maddock and Coller[27] who point out that these parts comprise 65% of the skin area, a much greater proportion than is generally realised. They found that a much greater shift of blood occurs to the extremities than to the Head and Trunk, the toes receiving most of all. The application of hot water to the arm has been found to increase the local circulation rate by as much as 4 to 8 times, with accompanying/

accompanying less marked changes in the blood flow of the opposite arm, the latter being influenced also by room temperature and the warmth of the individual [28]. All types of artificial fever except that due to foreign proteins, cause a marked increase in the pulse volume changes of the fingers, [29] and plethysmographic studies show that the tendency of the hands and feet to swell in warm weather is due to increased blood flow. [27]

This dilatation of cutaneous vessels even though accompanied by vaso-constriction in more central organs probably causes an increase in the total capacity of the circulatory system which may be the main cause of the increase in blood volume which occurs [3]. To prevent the ratio of blood volume to the capacity of the circulation from falling serum is added to the blood chiefly from the Liver [20] and the muscles, the water content of which has found to be decreased under these circumstances [3] and water is given up to the serum by the red blood corpuscles.

Peripheral vaso-dilatation despite a fall in arterial blood pressure results in a local rise of capillary pressure, but in the central organs the combination of the fall in arterial pressure and some vaso-constriction should lower capillary pressure and attraction of fluid by unbalanced osmotic pressure may occur. To conserve fluid the Kidneys secrete a more concentrated/

concentrated urine. Nishibori[30] suggests that this hydraemia may increase the ability to secrete sweat and may be the stimulus to thermal sweating. He found that a fall in the osmotic pressure of the blood accelerates the ability to sweat, and a rise in osmotic pressure suppresses it, whilst mere increase in the amount of blood has no effect.

PERSPIRATION AND SWEATING: In the above brief account of the main physiological reactions to a need for heat loss a distinction was made between surface moisture, and visible sweating. By surface moisture or insensible perspiration, is meant the escape of water, probably a transudate from blood vessels, by osmosis through the skin a process in which the sweat glands at least of the greater part of the body are apparently concerned little if at all. Martin[21] refers to this process as "the imbibition of water by the epidermis from the moist cutis", whilst Pembrey[2] points out that the process shows variations which would be expected in a transudation, being proportional to the temperature - greater if the air is dry and less if it is stagnant - and being diminished if the skin is clothed in fat. An increase in the blood flow in the cutaneous capillaries and a rise in skin temperature also increase the amount of insensible perspiration. Investigating the changes in the process with the moisture/

moisture of the air, Adachi & Ito[31] found that the cutaneous insensible perspiration varies in proportion to the changes in the loss of water from the Respiratory passages. Thus it gradually increases on breathing wet air but on breathing dry air it is gradually decreased but not enough to cover the increase in respiratory perspiration, so that the total loss is somewhat greater. Bazett[3] extends the term to include not only the evaporation of such a transudate from the tissue fluids or from the blood itself, but also that of fluid from the sweat glands when the rate of evaporation is greater than that of secretion, so that no visible collection of fluid occurs, which as Kuno[32] observes would make a greater contribution to insensible perspiration in dry moving air, or air at the low atmospheric pressure of high altitudes.
Kuno[32] [33] found that in insensible perspiration, in which he includes the discharge of moisture from the respiratory passages, the greatest amount of water is discharged from the palms and soles where the sweat glands are most dense, and he concludes that cutaneous insensible perspiration is due to two different processes - the physical discharge of water through the epidermis occurring all over the body surface, whilst the perpetual secretion of sweat makes an additional contribution in some minor parts such as the palms and sole. Although it must not be assumed that because/

because no sweat is obvious to the naked eye, no secretion is occurring,[21] an indication of how small may be the contribution of the sweat glands to insensible perspiration is afforded by the observation of Richardson[34] that in a case of congenital ectodermal defect the complete absence of sweat glands did not prevent the liberation when the subject was at rest of the normal quantity of water vapour of which 70% came from the skin and only 30% from the Respiratory passages. Keeton[35] states that a certain amount of vaporisation from the skin takes place in rabbits which have no sweat glands. Although the loss of heat by evaporation from the upper respiratory passages is almost the sole means of cooling such active animals as dogs, kangaroos and rabbits, it is not of great importance in sweating animals. In man exposure to heat increases the respiratory rate and pulmonary ventilation but Martin[21] states that although the amount of heat lost by this route is not inconsiderable it does not account for more than about 5% of the heat dissipated. Its importance may be enhanced when elimination from the skin is impaired by ichthyosis or absence of sweat glands, and MacLeod[36] points out that more rapid breathing also facilitates loss of heat by conduction from the mucous membranes of the Tongue, mouth and upper respiratory passages.

Turning/

Turning now to the secretion of the sweat glands, the process which we are more accustomed to think of as "sweating" or as "perspiration" (a "genteel" term which might well be reserved from the physiological point of view for the transudatory process which has been described above) the experiments of Kuno (32) (33) must be briefly considered. He made careful observations on the discharge of sweat, usually by passing a dry air current through celluloid dishes fixed to the skin, and thence into tubes containing calcium chloride which were weighed every five minutes. Skin and rectal temperatures during the observations were determined by the thermocouple method. These experiments yielded much interesting information and enabled Kuno to differentiate "thermal" from "mental" sweating. In normal subjects sweating in response to high environmental temperature began about thirty minutes after the rise of room temperature, and increased rapidly. In the summer time, or in abnormal subjects such as heavy drinkers, sweat was produced immediately after exposure to heat. Although the amount of sweating on different parts of the skin varied, the time of outbreak and the subsequent changes were much the same. Unless the heating was confined to a small part of the skin surface, when, possibly owing to increased excitability of the sweat glands/

glands due to direct heating, sweat appeared rather earlier on the heated region than on the other parts, the sweating occurred simultaneously all over the body surface except on the palms and soles, where the sweat glands have the characteristic of permanent secretion, and react to mental and sensory stimuli, but not to a rise of environmental temperature unless of extreme degree. The effective stimuli for these glands include pain of various kinds, and unusual sensations such as a desire to urinate, or the application of an Esmarch's bandage, and also strenuous exercise[(37)]. The most effective stimulus is mental stress, and mental arithmetic caused sweating associated with an increase in capillary blood flow and skin temperature in the palms and soles only, not being a strong enough stimulus to cause sweating on the Forehead or in the axilla, where sweating also occurs from mental stress. At fairly high environmental temperatures mental stimuli may cause general sweating as well as sweating on the palms and soles. Kuno considers that this mental sweating may be a phenomenon transmitted from animal life. In animals mental stress is usually followed by the need for muscular action, and, especially in climbing animals, moistening of the pads which increases the friction between them and the surface of the ground or of trees is of assistance in flight/

flight or fight. In man too, such increase of friction helps in grasping objects, and renders the sense of touch more acute, and it is of course often aided by voluntary moistening of the palms. This suggestion is further borne out by the fact that this mental sweating in man appears most profusely on the parts of the palm which would be in close contact with an object grasped or on the sole where it would touch the ground in standing. The Japanese language recognises the existence of this mental sweating in the expression "with sweat in the clenched hands", which denotes "with suppressed excitement" or "with breathless interest"; and the British soldier too perhaps acknowledges the phenomenon so far as the forehead is concerned in his expression "sweating on the top line" meaning in a state of anxiety over such matters as promotion.

The permanent secretion of sweat on the palms and soles is of further importance in the protection of the skin of these parts which might otherwise become dry and flexible. Both thermal and mental sweating occurs in the axilla, and Ogata[38] suggests that here mental sweating may be a function of the "apocrine" glands whilst thermal sweating is a product of the "eccrine" glands. "Apocrine" is the term applied to glands resembling sweat glands, but larger in diameter which show/

show various differences from the ordinary "eccrine" or "exocrine" glands in their structure, method of secretion and in anatomical distribution being chiefly found in the axillae and groins.(32 (39) Ogata noted that the axillary sweat glands of children respond to thermal agents only, whereas those of adults respond to mental as well as to thermal stimuli. Both apocrine and eccrine glands are present in the axilla, the former developing at a later stage of childhood.

As regards the local variations in thermal sweating already alluded to, Kuno found that sweating was most marked on the Head and Neck, and some areas of the anterior and posterior surfaces of the Trunk, the dorsal region of the hands and adjacent parts of the forearms. The armpits and groins sweat least and the apparent moistness of these parts is due to the fact that such sweat as is secreted there does not readily evaporate. A convincing demonstration of this is afforded by a man playing the bagpipes, in whom the armpit of the arm which holds the bag and is thus kept away from the side is quite dry, whilst the other armpit may be wet with sweat, as this arm must be held in close apposition to the side, in order that the hand may reach the lower part of the chanter. If the heating is more intense sweating may also appear on the palms and soles and axillary sweating may often become profuse. Sweating is slight on such parts of the/

the body as are clothed in fat such as the cheeks, the gluteal and mammary regions, and, as the cooling effect on the whole body of evaporation in these parts is slight, the avoidance of free sweating there is reasonable. All the areas where sweating is greatest are however advantageous for the elimination of heat, and regulation of local temperature in these areas is more important than in the extremities, since in the head and neck and trunk the superficial area is small compared to the mass and there are important organs in their cavities.

The secretion of sweat is under the control of the nervous system, the sweat glands being innervated by sympathetic fibres and the nervous mechanism may be called into play by central or peripheral stimuli, or by reflex action. The impulses are probably chemically transmitted by acetyl choline liberated at the nerve endings which acts upon the sweat glands,[40] [41] and the discharge of sweat is assisted by periodical contractions of the muscular fibres of the sweat glands[42]. Sweat centres in the brain stem have been described from time to time and Kuno[32] says that there is evidence that the sweat centres are situated widely in the cerebral cortex, the hypothalamic region, the medulla, and the spinal cord, and suggests that thermal sweating may be under the control of a centre in/

in the hypothalamic region and mental sweating under the control of a cortical centre. Sweating is prominent amongst the phenomena recorded by Cushing[43] following intraventricular administration of Pituitrin. He records nausea and vomiting, salivation, intense flushing, a marked fall of body temperature and sweating so profuse that the body is bathed in sweat. These effects are quite different from those caused by intravenous or intramuscular administration of this substance and are, he suggests, due to direct stimulation of the subependymal hypothalamic nuclei. The same effects are produced by intraventricular injection of pilocarpine whilst atropine given subcutaneously or previously introduced into the ventricle counteracts them. The absence of any such effect from adrenaline, and the sudorific action of pilocarpine bears out the observations of Foerster and Hasama quoted by Cushing that there is a parasympathetic as well as a sympathetic mechanism for sweating. It is possible therefore that sweating may be due to a direct effect of pituitrin on subthalamic sweat centres, and it is interesting too that Cushing observes that a substance resembling posterior lobe extract is found in the normal ventricular and cisternal fluids, and that it is increased by, amongst other things, emotional stimuli, suggesting a possible mechanism for the production of mental sweating. The sweat centres are also excited by asphyxia,/

asphyxia, a rise in carbon dioxide or a fall in Oxygen in the blood, which may be a subsidiary stimulus for the sweating which occurs with muscular exercise, and the reason why in severe exercise the sweating lasts long after the exercise has stopped[37]. At high temperatures and in muscular activity there is increased excretion of lactic acid in sweat and Fishberg and Bierman[44] suggest that lactic acid too may be a stimulus to sweat gland activity. Hydraemia with lowered osmotic pressure of the blood, already mentioned as a stimulus to sweating, may contribute to the more profuse sweating which occurs in summer, when the sweating centres are more responsive and the sweat glands are in what Kuno calls the "trained" condition. Although a high skin temperature may increase the excitability of the sweat glands,[45] the secretion is not dependent on the internal temperature and can be produced by warming the carotid blood when the temperature of the general body is unchanged. Iwatake[37] found that the sweating due to muscular exercise, which is of the same type as that due to heat, occurred sometimes before the rise in rectal temperature and sometimes when the rectal remperature showed an initial fall, and that on the cessation of exercise the secretion of sweat subsided much more quickly than did the temperature. Kuno made similar observations/

observations in connection with thermal sweating which subsided rapidly on return to cool air whilst the rectal temperature was still very high. The sweat glands can react directly to heat[46] but the intensity of the heat necessary to excite them is so high as to be unlikely to occur in daily life, and general sweating from reflex action may occur long before such local heating directly excites the sweat glands of the part. Cooling has the opposite effect and suppresses sweating and Ogata[47] notes a decrease in oxygen consumption following the intake of cold water at high temperatures. The suppression of sweating due to cooling was attributed by Hill[48] to cooling of the blood, as he noted that the suppression due to cooling a limb did not occur when the blood supply of the limb was occluded. Nishibori[45] also, noted that obstruction of the circulation of a cooled hand caused sweating on the general surface to increase, whilst restoration of the circulation caused sweating to decrease. Similarly obstruction of the circulation of a heated hand caused diminished sweating and its restoration a remarkable increase. On obstruction and restoration of the circulation the rectal temperature changed only very gradually, and no noticeable changes were recorded when sweating was depressed or augmented. Kuno, however, finds that some suppression does occur even when/

when the blood supply of a cooled limb is occluded, and concludes that cold may suppress sweating in a two fold manner - by a nervous reflex as well as by some sort of diminution in responsiveness of the sweat centres due to cooling of the blood. The subsidence of sweating on exposure to cold, even when the body temperature remains high indicates that sweating is essentially a preventive mechanism, effective only when the factors raising body temperature are in operation, and is thus useful in preventing a rise of body temperature but not in reducing an increased body temperature. Although usually associated with increased blood flow in the skin capillaries, sweating is not entirely dependent upon this, and sweating in the leg may be observed in association with vaso-constriction on stimulation of the sciatic nerve,[(18)] [(41)] and the sweating of syncope, fear and death, is associated with pallor and coldness of the skin. Nevertheless Kuno observes that whilst a moderate secretion of sweat can be kept going for 20 minutes or more without the blood supply, the glands can act completely only when the blood supply is sufficient. Ichihashi and Ogata[(49)] suggest that the diminution in sweating in a limb with occluded blood supply is due to lack of oxygen and, the rate of oxygen consumption of sweat glands being low, the oxygen contained in the neighbouring/

neighbouring blood or absorbed from the skin is enough to keep their function going for some time. A possible reflex effect of cutaneous vaso dilatation on sweating is suggested by Kuno[32] who noticed changes in sweating due to posture, such as sweating on one side of the body when the subject lies on the other side. He suggests that dilatation of blood vessels may mechanically stimulate some sensory nerves which elicit a sweat reflex, congestion of one side of the body causing the sweat reflex on the other. If this is so it is a further example of the economy adopted by the body in connection with sweating, for in the case of a man lying in bed, cooling by evaporation of sweat can take place only from the upper surface of the body, and not from the parts in contact with a sweat soaked in bed.

Mental unlike thermal sweating usually occurs at once in response to any causal agent, which suggests that the apparatus concerned is more sensitive, and its sensitivity too is reduced by cold. As already stated, mental sweating may appear on the general body surface when the environmental temperature is high. At still higher temperatures mental stimuli may cause sweating only on the general body surface, and not on the palms and soles, whilst when the heat is still more severe, these stimuli inhibit sweating on the general/

general body surface and in this case palmar sweating occurs normally or may be slightly decreased. These paradoxical phenomena observed by Kuno are hard to explain, but he suggests that they indicate some connection between the highest nervous centres, and the sweat centres. Some cortical centres may have the power of exciting or inhibiting the sweat centres, the exciting effect being as a rule predominant, but the inhibitory becoming effective when the sweat centres have been vigorously excited.

The observations cited above, and the fact that sweating is usually found to be suppressed or restored concomitantly over the whole body surface[50] indicate that the secretion is controlled by nervous centres, and not by any local process in the sweat glands themselves, although it may be remarked that Ichihashi and Ogata[51] noted a varying degree of susceptibility in different glands illustrated by the variations in the number of sweat glands excited by different stimuli.

To sum up - perspiration is a transudation influenced chiefly by the vascularity and temperature of the skin and slightly by the humidity of the air, and to it the sweat glands make only a small contribution. Sweating is an active secretory process controlled by the nervous system, and may be brought about/

about by a rise in blood temperature or by a nervous reflex, the afferent path being temperature fibres and other afferent nerves, whilst efferent fibres pass to all sweat glands from cerebral sweat centres. The chief stimuli to the sweat centres are a rise in environmental temperature, mental stress, and various sensory stimuli, asphyxia, and lowered osmotic pressure of the blood.

The important part in temperature regulation played by the evaporation of sweat is well illustrated by cases which have been described of by congenital ecto-dermal defect, in which there is complete inability to sweat due to absence of sweat glands, and usually also deficiency of scalp and axillary hair, and partial absence or incomplete development of teeth.[2][34][52][53] The evaporation of cutaneous moisture in such cases was enough to keep the body temperature within normal limits even on the performance of moderate exercise, though here the body temperature rose somewhat higher than is beneficial. Martin[21] calculated from the observations of Loewy and Wechselman that a loss of about 0.18 calories per minute may result in such cases from evaporation of insensible perspiration. On exposure to external heat however, there could be no increase in water to be evaporated and the body temperature rose sharply despite normal cutaneous vaso-dilatation. The heat loss by radiation, convection, and/

and conduction, proving insufficient to maintain the temperature within normal limits, the subject had to soak his shirt with water, and so obtain the effect of evaporation. The habit of the elephant which has few sweat glands, of spraying himself with water from his trunk is undoubtedly directed more to temperature regulation than to the desire for cleanliness with which the animal is often credited, and when a source of water is lacking he will often insert his trunk in his mouth and spray himself with saliva.

The importance of the sweat glands to the horse will not be doubted by anyone who has had the misfortune to own in India a horse suffering from the non-sweating syndrome or "dry coat" in which the animal is unable to remain in the plains in hot weather on account of its inability to regulate its temperature and consequent liability to develope Heatstroke.[54] (See Appendix I). Whilst married officers have been known to acquiesce without undue emotion in the annual hot weather visits to the hills of their wives and families, few can regard with equanimity a similar visit on the part of their polo pony.

It is obvious that when the environmental temperature is higher than that of the body no heat can be lost by conduction, convection, or radiation, and in fact heat may be gained by the latter process, so that the ability to sweat and so to lose heat by evaporation/

evaporation is essential to men living in hot climates. Alcohol and the after effects of illness may cause a tendency to sweat easily, and a tendency to copious sweating is well known in such conditions as Tuberculosis and Hyperthyroidism, whilst hyperidrosis, generalised or localised, apart from any other disease is not uncommon, and often occurs in neurotic individuals. Normal healthy individuals show marked differences in their ability to sweat freely and such individual variations in capacity for sweating accounted for the fact recorded by Hill [55] that in Mesopotamia in 1917 when the temperature rose in stages, groups of cases of Heatstroke occurred at 105°, 110°, 115°, and 120° F. Differences also occur in the same subject and Ichihashi and Ogata [51] found that the amount of sweat in 24 hours varies markedly from day to day in summer, and that the variations are chiefly connected with food intake, whereas the intake of water or output of urine have little effect. Whilst this increase in temperate climates is probably due to an increase in insensible perspiration from an increase in metabolism, it is in hot climates chiefly due to a sweat reflex which they believe is elicited by the intake of food when the ability to sweat has been elevated on account of a high temperature. In general those who sweat less easily are less/

less suited to a tropical life, but excessive sweating is also undesirable and Bazett[3] notes that those acclimatised to the Tropics sweat much less than new-comers and so avoid a wasteful dehydration, and other adverse effects which will be described later. Too much sweating is not only useless, but may even impede heat loss by soaking the clothes. In the acclimatised subject the response of the nervous apparatus is controlled by training and any excessive excitation is avoided.

The ideal subject for tropical life would be one who in a sedentary occupation does not sweat at all in a cold or temperate season, but does so in summer but not ~~soo~~ too profusely.

In view of these variations in sweating it is not surprising that there are considerable differences in the various estimates which have been made of the amount secreted in 24 hours. c.c.

McSwiney[56] finds that 600 to 700 cc are under normal conditions evaporated from the skin daily, and an average of many estimates quoted by Kuno[32] would be about 3 to 4 kilogrammes per day. Hunt[57] found that the loss requiring replacement amounted in Europeans in the Deccan in hot weather to as much as 3 gallons and that under unfavourable conditions the daily loss of fluid may amount to 30 pints, whilst MacEwen[58] considers that under moderately favourable conditions/

650

3500

13,00

17,000

conditions with light exercise the loss is about 12 pints. These apparently discordant results are of course due to the different climatic conditions under which the secretion of sweat has been studied, as well as, possibly to individual variations in sweating. Adolph[59] finds that the rate of sweat production is probably directly proportional to the "effective temperature" above 90°F. The exact amount of sweat produced daily is of less importance in the avoidance of heatstroke than the ability to sweat when necessary, and also what Ito and Yabuki[60] refer to as "effective sweating" - that is the amount of sweat which evaporates per unit of time from the body of a subject sweating profusely. Under the most favourable conditions this may approximately correspond to the maximal amount of sweat produced which is about 0.8 to 1.01 litres per square metre per hour or between 1 and 2 litres for the whole body, though Moss[61] recorded in miners losses up to more than 2½ litres.

As an indication of the amount of fluid requiring replacement on account of sweating alone the foregoing figures may be summarised as follows, and in terms of pints since that is the measure we are perhaps most accustomed to deal with in this connection. In temperate climates with little exertion the amount of sweat in 24 hours is about 1 or 2 pints, whilst with the/

the ordinary exertion incidental to say a soldier's life, it may amount to some 5 to 6 pints. Hard work in conditions of high temperature and low humidity will cause a loss of from 10 or 12 pints up to as much as 25 to 30 pints in 24 hours, the maximum loss per hour during spells of work being in the neighourhood of 2 to 4 pints.

The figures given by various writers for the chemical composition of sweat also show considerable differences which are largely due to the unavoidable admixture of secretion from the sebaceous glands,[62] and to variations in the composition of sweat with the duration and the region of the sweating.[59] [63] Divergence of opinion on the pH of sweat is due to the readiness with which sweat is decomposed by the bacteria normally present in it,[56] but sweat is normally acid, though after prolonged sweating it may be neutral and later alkaline.[62]

In general sweat consists of about 99% of water, and sodium chloride accounts for not less than ¾ of its solid constituents, most of the remaining quarter being urea. Potassium chloride, glucose, uric acid, lactic acid, phosphates, and creatinine are present in small amounts.[3] [19] [41] [62] McSwiney,[56] observing that the constituents are very variable gives the following average figures.

Subject	pH	NH_3N	Urea	Amino Acid Nitrogen	Glucose	Sodium Chloride Grammes per 1000 cc
		Milligrammes per 100 cc				
Female	6.57	6.0	19.3	6.5	20.0	3.00
Male	6.14	4.7	21.44	5	12.6	3.70

The amount of urea may be increased in renal disease[(19) (33)] when the sweat may also be more concentrated, whilst after severe exercise lactic acid is more often to be found in the sweat than in the urine.[(11)] Its excretion by this route may be an important means of getting rid of lactic acid from the body, and so protecting the Kidney from damage, facilitating muscular work and causing the fall in Hydrogen ion concentration during the achievement of "second wind".

Marsh[(64)] suggests that in circumstances such as those in the Persian Gulf littoral where Europeans have to sweat continuously, and urine may be passed only once a day, some usurpation of Kidney function by the sweat glands may occur. The onset of sweating is associated with an increase in the output of CO_2 from the skin,[(2) (19) (41)] and this may be responsible in part at least for the fall in CO_2 content of venous blood in the skin which may occur before there is any fall/

fall in alveolar CO_2 tension (65).

Sweating has therefore some minor importance as an excretory process, and Iodine, Bromine, and Mercury when administered internally, may be excreted in sweat, whilst chromidrosis or the excretion of coloured sweat has been frequently reported, though this is commonly due to bacterial activity (66).

Sodium chloride, the most copious constituent of sweat, is also of the most importance in disturbances of heat regulation.

The figures for sodium chloride of many observers are in fairly close agreement with those of McSwiney[(56)] whilst others report higher concentrations, the figures ranging from about 1.8 to 9.5 grammes per 1000 cc. (33) (41) (44) (56) (59) (62) (67). The conflicting data are probably due to differences in the methods of collection, as well as to mixture with salts and other substances from the skin, such as broken down sweat cells or sebaceous matter. Under normal conditions the sodium chloride of sweat makes only a small contribution to the daily loss of sodium chloride which amounts to about 10 to 20 grammes per diem, (21) whereas the loss in the urine is nearly as great as the average intake.

Although the percentage of chloride in sweat has been found by some to increase during prolonged sweating/

sweating, (61) (68), Dill[69] and his co-workers found that in extreme dry heat as part of the process of acclimatisation, the concentration of chlorides in sweat decreases; an important adaptation in view of the grave results of excessive salt loss which will be described later. Whatever the exact concentration of sodium chloride in sweat, the secretion is markedly hypotonic, and so evaporates readily, and extracting from the body the latent heat necessary for its evaporation each gramme of water removes from 582 to 600 small calories of heat (21) (70) (71). In terms of pints it may be said that 1¾ pints absorb about 600 large calories, (a large calorie being equivalent to 1000 small calories).

The preservation of the blood volume when the capacity of the circulation is increased by vasodilatation has already been discussed, and the loss of large amounts of fluid by sweating calls for a similar adaptation. We have seen that as much as 1 or 2 litres may be lost in an hour, and yet no concentration of the blood may occur, even when no fluid is drunk, since the percentage haemoglobin has been found to be unchanged. Fluid is attracted by the raised osmotic pressure of the plasma proteins from the muscles which give up some 67.89% of the total water lost,[72] and from the tissue spaces whilst there may be too some ill understood action of the central nervous system upon/

upon the tissues.[4] Reserves of fluid can thus be mobilised to withstand the loss of even several litres of fluid from the skin but if the exposure to heat is prolonged it is necessary to replenish the reserves by copious drinking. The preservation of water in the body is aided by the Kidneys which excrete a more concentrated urine, the most important adaptation being probably a more complete reabsorption of water in the renal tubules, glomerular filtration being also reduced. The urine is usually alkaline, probably to counteract the alkalaemia which might result from the fall in alveolar and arterial CO_2 tension resulting from increased pulmonary ventilation.

According to Marriott[73] the average daily excretion of urea is 30 to 35 grammes a day, which requires for its excretion 650 to 800 cc of urine, but in subjects who abstain from food and water the urine volume may fall to 408 cc which amount is secreted even though dehydration is occurring. Lee and Mulder[63] found that in subjects who refrained from fluid intake for 12 hours a "basal level" of urinary volume of 15 to 30 cc per hour was reached during the ensuing three hours in a normal environment, whilst in a hot environment the "basal level" was lowered to 8 to 15 cc per hour. This reduction in volume they take to illustrate the limit of the concessions allowable to hydration/

hydration functions at the expense of excretory functions. For excretory purposes, then, urine must be excreted, and probably about 400 cc in 24 hours[20], and if continued loss of fluid by sweating occurs and water intake is not increased dehydration must occur.

FAILURE OF ADAPTATION TO HOT ENVIRONMENTS.

Mention has been made of such possible failures in heat regulation as excessive salt loss and dehydration, and these and other kinds of failure in adaptation to hot environments must now be considered.

CIRCULATORY INSUFFICIENCY.

It is in the first place evident that the circulatory system plays a prominent part in temperature regulation and that as Pembrey[2] says "a good Heart and vasomotor control are necessary for the flushing of the skin and the maintenance of an efficient pressure of blood". Men who suffer from cardio-vascular disorders or vasomotor instability are less suited to life in hot climates and Hill[55] observed that such people were the first to suffer from Heat-stroke in a spell of hot weather. The thorough physical examination conducted before embarkation ensures that soldiers who suffer from such conditions are not allowed to proceed to hot climates, but circulatory breakdowns may occur from various strains imposed/

imposed upon a normal system. Severe muscular exertion, especially if associated with the added emotional strain of competition, heavy meals or alcohol may, if indulged in at times when the circulatory system is already responding to the demands for heat loss, tax the system too highly and cause collapse. Muscular efficiency and alimentary functions may thus be impaired by anoxaemia, whilst the nervous system is usually affected, with resulting irritability, fatigue, faintness, or actual unconsciousness.

As the circulatory failure may to some extent reduce heat production whilst the anoxaemia acts as a stimulus to sweating one would not expect this type of failure to result in a high temperature. Some such mechanism is responsible for the type of case later to be described as Heat Exhaustion and in this condition the temperature is usually normal or subnormal. Another factor contributing to circulatory insufficiency in more severe degrees of heat is dehydration in which condition the fatty tissues the Brain, Heart and Skeleton lose relatively little water compared with the muscles and the blood. Whilst the muscles and the skin suffer little from dehydration, the process when it affects the blood is accompanied by impairment of the circulation, and secondary functional disturbances in all parts of the body.

DEHYDRATION.

It has already been pointed out that large amounts of water may be lost when heat regulation by the evaporation of sweat is essential, and that whilst much can be made good by the mobilization of the body reserves of fluid assisted by conservation of water by the Kidneys, a time may come when the body reserves are depleted, and when further compensation by the Kidneys secreting a more concentrated urine could only be attained at the expense of their excretory functions, and that then dehydration of the tissues must result. The subject of dehydration in heat regulation is closely associated with that of loss of sodium chloride, the chief constituent of sweat, but as somewhat different clinical states result from a predominance of one or other of these factors, they will be considered as far as possible, separately.

The critical level in water loss is, according to Lee[20], when the water content of the body reaches 20 to 25% of the body weight, the percentage to which MacCance[72] states the extracellular fluids of the body normally amount. Apart from salt loss most of the physiological results of dehydration are due to concentration of the blood, or anhydraemia, and the condition of the blood is a good index to the degree of dehydration as a whole.[73]

Moderate/

Moderate degrees of heat cause a decrease in the red cell count, haemoglobin content, and viscosity of the blood, and this hydraemia has been described above as an accompaniment of the early reactions to heat. The phase of blood dilution continues until sweating becomes severe enough to swing the balance towards anhydraemia. The statement that considerable loss of fluid may result in no blood concentration should be qualified by the remark that if the loss is a sudden one an immediate concentration of the blood of 15% or so may result.[(73)] Water is later given up by the reserves and the blood returns to its normal composition if the loss is not continued. When water is given after dehydration an immediate dilution with an abrupt fall in haemoglobin concentration occurs, after which the blood concentration gradually increases as the excess of water is excreted by the Kidneys and taken up by the dessicated tissues. Equilibrium is ultimately reached if water continues to be administered but the replacement of the body reserves of fluid, if they have been extensively drawn upon seems to take many hours.[(57)] In dehydration the red cell count increases, and the serum proteins may increase over 50% in concentration, an increase of as much as 100% having been recorded[(73)]. The viscosity of the blood is consequently increased, and marked increase in viscosity/

viscosity in the dehydration due to loss of intestinal juice is recorded by Herrin.[74] Excretion of inorganic salts in the urine may maintain the salt concentration of the plasma within normal limits, but it may be raised in sudden anhydraemia or when renal function is impaired. In considerable degrees of anhydraemia the amounts of urea, non-protein nitrogen, uric acid, sugar, and lactic acid in the blood may all be raised, and acetone, aceto-acetic acid, and oxybutyric acid may be present. The oliguria associated with dehydration could well account for a rise in some of these substances and MacCance[72] observes that the plasma chlorides, which may be reduced in this condition tend to move inversely with the blood sugar. The rise in blood urea however, he points out, may occur whilst renal function is still relatively normal, and a possible explanation for the rise in cases associated with salt deficiency may be the reabsorption of urea to raise the subnormal osmotic pressure of the plasma. Mackay[75] found that in dehydrated rabbits the blood urea concentration rose very high, and he attributed the rise mainly to increased urea formation from accelerated tissue catabolism. The rate of urea excretion always gradually increased, although the urine urea never attained a fixed or constant maximum. An increase in blood urea and uric acid in children with clinical evidence/

evidence of dehydration was observed by Mitchell and Jonas[76] only when marked toxic symptoms were present. They found a decrease in the CO_2 capacity of the blood plasma in such cases only when hypernoea was a feature, whilst Marriott[73] found a decrease in the oxygen capacity of the haemoglobin in such cases.

Various alterations in the acid-base balance have been recorded, though Lee[20] found it to be little affected unless heating is very extreme and a breakdown occurring from some other cause.

In 168 cases of dehydration due to infantile diarrhoea studied by Maizels and Smith[77] the alkali reserve of the blood was less than one half of the normal, moderate acidosis being present in 42%, normal plasma bicarbonate in 33% whilst 16% shewed an alkalosis.

The plasma bicarbonate and chloride they found vary inversely, and whilst loss of chlorides in the stools may be so great as to lead to deficiency of chlorides in the blood, it is more usual for the accompanying loss of water to be so much more marked that the blood volume is reduced and the chloride content of the blood is actually increased. Thus the greater the dehydration the greater is the plasma chloride, and the less the alkali reserve. Dehydration due to sweating similarly raises the plasma chloride concentration; the maintenance of a normal or above normal/

normal level of chlorides in the blood being, as McCord[62] points out, due partly to concentration of the blood and partly to withdrawals of chloride from cellular tissues, especially from the red blood corpuscles. As profuse sweating continues there is a sudden decline in gastric acidity and a general state of acidosis supervenes.[62] [78] The Hydrogen ion concentration of the blood is increased together with its lipoidal content, and the alkali reserve, plasma bicarbonate and CO_2 tension of alveolar air are diminished. An increased alkali tolerance is shown by Sellard's test in which, in this condition, large amounts of alkali can be ingested without causing a change in the urine towards alkalinity.

The urine is very acid, as the Kidneys must play their usual part in regulating the pH of the plasma, but this function may fail in severe dehydration or salt deficiency, and with the failure of ammonia formation an intensely acid urine is secreted and the ammonia coefficient is not raised.

Functional impairment of the Kidneys may also be evidenced by diminution in the amount of urine which may show traces of albumin, casts, and a small amount of sugar, as well as an increase in organic acids. Various reasons for this renal failure are suggested by MacCance[72] who points out that the volume of urine secreted daily is probably controlled by humoral factors/

factors, and by the Pituitary, and that after mild loss of fluid by sweating, or reduced water intake, the volume of urine may be very small indeed without any evidence of a reduction in the rate of glomerular filtration, whilst conversely the daily excretion of water may be normal or excessive when glomerular filtration, and the power to excrete urea and creatinine are certainly reduced. In dehydration the reduction in the blood volume may result in only a proportion of the normal number of glomeruli being filled, so that the number of active glomeruli is reduced, and we have already seen that in water deprivation or at very high temperatures the daily volume of urine may be very small. The volume of blood flow through the Kidneys is accepted as being probably of more importance in the secretion of urine than the blood pressure, a fall of which should not affect the volume of urine or the concentrating power of the tubules, provided they remain adequately nourished. The increase in plasma proteins causes a rise in the colloidal osmotic pressure of the plasma, which would reduce glomerular filtration unless the arterial blood pressure rises in compensation. The increased viscosity of the blood may decrease the rate of the circulation through the Kidney and so reduce its efficiency. (21) (72) (73) (79). A further suggestion as to the cause of the renal failure/

failure is that the Kidney cells may be specifically damaged by salt withdrawal (72).

The usual mechanisms then, for controlling acidaemia are brought into action in these cases, and when the alkali reserve is reduced, pulmonary ventilation is increased, the alveolar CO_2 falls, and the acids and the ammonia of the urine are increased. A subsidiary mechanism is suggested by Fishberg[(44)] who notes that base economy is shown by the ability of the body to excrete sweat at a much lower pH than that of the blood plasma, and that the excretion of lactic acid and lactate spares fixed base, as a considerable amount of lactic acid thus leaves the body unionized. A factor contributing to the tendency to acidaemia may be overproduction of acids in the tissues due to suboxidation associated with a diminished flow of blood.[(73)] This diminution is due not only to the increased viscosity of the blood but to a reduction in the blood volume, sometimes by as much as 40% or more of the total. A compensatory constriction of peripheral arterioles results and the volume of blood flowing through certain parts of the body is greatly diminished just as it may be in shock or haemorrhage. The importance of this reduction in blood volume has been emphasised by Atchley[(80)] in the variety of medical shock which he refers to as "anhydraemic" as opposed to "toxaemic" shock. In dehydration the blood flow through the extremities/

extremities may be reduced to less than 10% of the normal flow.

Dehydration must obviously reduce the power of temperature regulation since the water available for evaporation is diminished and may soon become insufficient. The reduction in blood flow through the extremities is also important, for they normally receive a much greater amount of blood than the rest of the body in response to a need for heat loss.(27) At temperatures much higher than that of the blood, when any heat exchange by radiation or convection must be in the direction of a gain in heat by the body, reduction in peripheral blood flow may, as Barbour and Tolstoi(81) point out, have some effect in preventing the body from acquiring too much heat, but any such effect must be trivial in comparison with the interference with heat loss by evaporation.

SALT LOSS.

Sweat, although strongly hypotonic, can remove considerable quantities of sodium chloride from the body, and Moss [61] found that when sweating was profuse the chloride loss may be very great and might exceed the loss in the urine. Whilst the initial effect of loss of sweat is to raise the chloride concentration of the blood, yet when water is drunk to replace the sweat, and to restore the blood volume, the body fluids tend to develop a lower chloride concentration. This is met by liberation of chloride from the body stores, and, when these are exhausted and the subject continues to drink water greedily, as those in this state of salt depletion will, the body, in order to correct the resulting hypotonicity, gets rid of the surplus water by means of the Kidneys and also sometimes by a burst of sweating. The blood volume then rapidly falls to its former low level or becomes even lower since more salt must be lost in the sweating, and thus a vicious circle is established, which can be broken only be the ingestion of salt.[82] Diarrhoea and vomiting may add to the chloride deficiency.

Estimations of the amount of sodium chloride which may be lost vary slightly, as do the figures given for the average amounts of sweat lost and for the average/

average chloride concentration of sweat. Martin[21] estimates the daily loss of sodium chloride from all sources as 10 to 20 grammes.

McCord[62] assuming that the soldier may lose 3000 cc of sweat with a reasonable average of 6.5 mg per cc of sodium chloride puts the daily loss from sweat alone at about 20 grammes, and quotes Marschak and Klaus as stating that the longer the sweating the higher the chloride concentration of sweat whilst the intake of water alone increases the output of water and of minerals. Kuno[32] taking average low figures estimates the loss of sodium chloride in sweat as 16 grammes per diem from which figure McEwen,[58] pointing out that an additional 12 grammes are lost in the urine[83] and that about 4 grammes per diem are necessary for bodily metabolism, calculates that the total requiring replacement is not less than 32 grammes under normal conditions, but that under unfavourable conditions of exposure and exertion or reduced salt intake it might amount to double that amount.

Hancock Whitehouse and Haldane[84] found that individuals differ not only in their ability to sweat but in the salt content of their sweat. They found that the percentage of sodium chloride rose with prolonged sweating, and attributed this to exhaustion of the sweat glands since less work is involved in separating from/

from the blood plasma containing fully 0.7% of sodium chloride, a liquid containing over 0.3% of sodium chloride than in separating a much more dilute solution. They agreed, however, with many other observers that acclimatised people are probably capable of prolonged moderate sweating without much loss of sodium chloride. Talbott[85] noted that when, in the earlier days of exposure to hot environments the loss of chlorides in sweat is increased, the urinary excretion of chlorides is diminished. When, however, good adjustment to the conditions is achieved, the salt concentration of sweat decreases and the amount of chlorides in the urine increases. This is in accordance with Dill's[69] observation that if there has been an opportunity for acclimatisation the sweat is so dilute that 10 litres a day may be lost without the need for an abnormal salt intake, and that except under severe conditions an intake of 15 grammes a day is adequate. There is, however, no doubt that, in unacclimatised subjects at least, continued sweating, combined with copious drinking of water without added salt, can lower the chloride concentration of the body fluids to dangerous levels, and Lee[20] finds that the danger point at which cramps may occur is when the serum chloride concentration reaches 100 mille equivalents per litre, which corresponds/

corresponds to 365 milligrammes per 100 cc. Talbott and Michelson[86] believe that the critical level of chlorides depends largely upon individual susceptibility, acclimatisation, and the length of the prodromal period before the onset of cramps, and Atchley[87] [88] points out that, though tissue chloride appears to change in the same direction as serum chloride, one must be guarded in drawing conclusions as to total chloride metabolism from changes in its concentration in the blood serum, which may be higher following a period of actual negative balance. Practically all the changes occur in the urinary constituents, and Lee states that for safety the daily excretion of sodium chloride should not be allowed to fall below three grammes. Maizels and Smith[77] whilst agreeing that if chloride is present in the urine the blood chloride is probably not low, point out that it may be absent from the urine when the blood chlorides are low or normal, or even, if the Kidney is so damaged as to fail to excrete chlorides, when there is a considerable excess of chlorides in the blood.

The occurrence of painful cramps especially in the calves, forearms and abdominal muscles in workers in hot environments such as stokeholds and deep mines has long been recognised, and Talbott[89] in a historical survey of the condition cites many references to it from/

from 1878 onwards. The term "Heat Cramps" has more or less replaced such names as "Stokers" or "Miners Cramps". The term "Morbus Brittanicus" recently suggested by Kofoed [90] seems to be not only unnecessary, but, by emphasising its prevalence amongst British Stokers, even misleading, since Elliott [91] found it to be more common in negro than in white stokers. Moss [61] in 1923, describing the condition as due to a combination of high air temperature, excessive drinking of water, and continued muscular work, said that Professor J.S. Haldane had suggested that it was caused by loss of chlorides. The administration of salt prevented the cramps and also all other bad effects of working in high temperatures, the men feeling much better both at work and afterwards.

Similar good results were obtained in 1908 by Elliott [91] but he does not seem to have suspected the cause of his success, as he writes that "the hot bath may be tried but it acts slower and is not so sure as the rectal injection" of hot saline. Replacement of the salt loss by additions of salt to the diet, or the drinking of salt solutions is now an accepted prophylactic measure in occupations in which heat cramps are liable to occur (69) (86) (89) (92) to (97).

McEwen [58] believes that salt depletion in lesser degree is a frequent and often unrecognised source of ill/

ill-health in hot climates, and attributes to this cause such symptoms as undue fatigue, lassitude, headache, sleeplessness, and lack of concentration. Ill effects of salt deprivation mentioned by Cannon[98] and Winton and Bayliss[99] are early loss of appetite, tiredness, muscular soreness and stiffness, sleeplessness, muscular twitchings, and excessive sweating. The excessive sweating, which McEwen[58] also finds to be a symptom of salt deficiency, may, he thinks, be followed by such minor disorders as "prickly heat" or cramps, or by more severe ones such as Heat Exhaustion. This excessive sweating may be due in part to the hydraemia accompanying the early stages of fluid loss, but there is evidence that certain degrees of salt depletion do encourage sweating whilst more marked depletion may have the reverse effect. Marsh[64] [82] considers that hypochloraemia is probably responsible for the anidrosis which often characterises prodromal and developed cases of Heatstroke, possibly because the sweat glands are unable to secrete sweat when the blood chlorides are low, or because a low blood chloride may induce a secondary reduction in blood volume which depresses sweating. Allen[100] suggests that depression of sweating may result from relative impermeability of the blood vessel walls due to the altered osmotic pressure resulting from salt loss. This would have the/

the effect of depressing sweating, since the sweat glands make sweat from lymph which has passed out of the blood vessels into the lymph spaces around the sweat glands. Walinski[101] prior to the therapeutic induction of physical hyperthermia gives 10 cc of a 20% solution of sodium chloride intravenously, which he finds inhibits sweating. This may be due to raising of the osmotic pressure of the blood, which as we have seen depresses sweating,[30] and later perhaps, in accordance with Allen's views, to the attraction of fluid from the tissue spaces, depriving the sweat glands of the material for making sweat. A supply of salt is evidently necessary for sweating and Talbert[67] who found no evidence of correlation between the chlorides of sweat and urine found that there was a correlation between the blood and sweat chlorides. Barbour[102] found that, although in the more severe environments causing early and abundant sweating the serum salt percentage is less increased than when the atmospheric cooling power is high, it is still increased and thus affords a continuous supply for sweating.

The blood lipoids, on the other hand, appear to cease mobilizing as sweating progresses, presumably because they contribute in some way to sebaceous secretion, for which, as it does not evaporate, there is/

is no continuous demand. That minor degrees of salt loss, on the other hand, encourage sweating is indicated by the observation that excessive sweating is amongst the phenomena due to exposure to heat which are relieved by the intake of salt solution. Ito and Kosuge[103] found that the excessive sweating which occurred in subjects in a room at a temperature of 46°C (dry bulb) and 36°C (wet bulb) was made more profuse by the intake of 0.8 to 2 litres of ordinary water. The general phenomena were, however, more gradual in onset than when water was withheld, the rise in temperature was slower, and the subjects could remain in the room for 125 to 160 minutes. When, however, 1.6 to 3 litres of a 1% solution of sodium chloride were given the symptoms were far less severe, the body temperature rose more gradually, and the heat could be borne for 170 to 215 minutes. Sweating at first increased rapidly but later decreased gradually. They concluded that the intake of salt solution suppresses excessive sweating on the general body surface and relieves hyperthermic phenomena. Kuno[32] and McCord[62] describe similar observations made in 1929 by Marschak, Klaus, and Dukelskaga who found that at a temperature of 46°C with a relative humidity of 50% men from whom water had been withheld showed concentration of the blood at the end of two hours. They suffered severe subjective phenomena of thirst, sleeplessness and headache,/

headache, and were unable to bear the heat for more than two hours. Drinking of water made things only a little better; sweating and secretion of urine were profuse, the temperature and pulse rate rose, but the general phenomena were less severe, and the heat could be borne for three hours. Drinking a 1% saline solution however eliminated all discomfort, there was no concentration of the blood, no excessive loss of water by sweat or urine, only a slight rise in temperature, and the men felt better and could endure the heat without difficulty for over three hours. They recommended the use of 0.5% sodium chloride solution, which they found promotes a sense of wellbeing and comfort, avoids cramps in muscles and in the intestinal tract, avoids deprivation of tissues of their normal content of minerals, and water, and prevents gastric hypoacidity. Kuno[(32)] draws attention also to the three stages in the elimination of water from the body described by Tobler. First to take place is the discharge of water independent of any other substance, which he calls "concentration water", next that in combination with salt or "reduction water", and finally that produced by destruction of tissues, "destruction water". The "reduction water" can only be restored by the simultaneous or previous supply of salt with the water.

The use of saline solutions in the prevention of heat/

heat effects, other than heat cramps, and in the treatment of such cases in which salt loss is predominant has been found most efficacious by many other observers (20) (32) (61) (62) (82) (86) (104) (105), and will be discussed later. In other conditions, too, when sodium chloride has been lost by the body, its replacement has been proved to be beneficial. Thus Kydd[106] reports good results from the administration of salt and water in diabetic acidosis, where salt and water is lost by the Kidneys and by vomiting. Administration of fluid with no salt is dangerous, as dilution occurs and the effects of salt depletion become even more apparent. The extreme hypochloraemia following the prolonged vomiting of high intestinal obstruction may cause death with symptoms of weakness, anorexia, oliguria and profound depression, and the surgeon recognising that the hypochloraemia is as important as the dehydration factor, replenishes the dehydrated tissues by giving sodium chloride to restore the depleted blood chlorides (107) to (114). Salt is of benefit also in cases of duodenal fistula[115] and hyperemesis gravidarum,[116] and in Typhoid, Smith[117] believes maintenance of the salt and water balance of the circulating body fluids to be as important as correct feeding. Barber and Oriel[118] found that the whole blood chlorides were diminished in acute and chronic allergy, and vary with exacerbations, and remissions/

remissions, but Black and Braden[119] recently reported that the blood chlorides both in allergic and in normal subjects show considerable daily variations, dependent upon humidity and barometric pressure, and not associated with allergic symptoms.

There is then much evidence that loss of sodium chloride by sweating or in other ways may gravely embarass normal metabolism, and the way in which it does so will now be considered.

Haldane[120] described heat cramps as being a form of water poisoning brought on by the combination of great loss of chloride by sweating, the excessive drinking of water, and temporary paralysis of renal excretion. Normally the tendency to a fall in the osmotic pressure of the plasma from loss of chlorides in sweat, and excessive water drinking, may be compensated to some extent by the kidney excreting the excess of water whilst retaining chloride. During hard muscular work, however, which Moss[61] found to be an essential causal factor in heat cramps, the kidneys are thrown out of action by the diversion of the extra blood supply to the muscles and skin[121] and the body is left defenceless against the loss of chloride. Haldane and his coworkers[84] [122] pointed out that the essential factor in the causation of the symptoms is not well expressed as a decrease in osmotic pressure,/

pressure, being rather an abnormal increase in the diffusion pressure of water, to the slightest increase or decrease in which the body is acutely sensitive. Priestley[123] suggested that the main regulation of blood volume by the Kidney is dependent on the diffusion pressure of water in the blood, and is modified by the inability of the Kidney to hold back water when the diffusion pressure of water in the urine is considerably below that of blood. Water intoxication produced by the forced administration of water has been studied experimentally, and some of the symptoms shewn by dogs in this condition resemble the symptoms ascribed by MacEwen[58] to salt depletion, and the symptoms of various clinical conditions due to heat. The symptoms in dogs described by Rowntree[124] were asthenia, restlessness, frequency of micturition, diarrhoea, nausea, retching, vomiting, salivation, tremor, muscle twitching, ataxia, convulsions, frothing at the mouth, stupor, and coma often ending in death. There is considerable blood dilution, but the concentration of chloride and sodium ions decreases to a much greater extent than would be expected from the degree of blood dilution (125) to (128). Although there may be a marked increase in the elimination of urinary chlorides[127] the decrease in blood chlorides is as marked in dogs with anuria or oliguria as in others[125]. Dogs which vomit earliest and most frequently/

frequently, thus losing chlorides become more rapidly toxic, and suffer from more marked tremors ataxia, prostration and convulsions.[129] Rowntree[130] amongst others attributes the symptoms to the raised intra-cranial pressure which is probably a manifestation of disturbance in the salt water equilibrium of the central nervous system. This rise of intracranial pressure may help to explain the very decided increase in blood pressure caused by giving large amounts of water to patients with hypertension[131]. Spectacular and almost immediate relief of the symptoms of water intoxication follows the giving of salt solution intravenously. The important factor in the mechanism of water intoxication is thought therefore to be neither retention of water in the body, nor blood dilution, but loss of chloride, and the resulting disturbance of the water: salt equilibrium of the body.

Talbott[89] does not support Haldane's[120] view that heat cramps are a form of water intoxication for he found no inability of the kidneys to excrete water and seldom any decrease in the osmotic pressure of the serum, but the chief aetiological factor is the same, namely the lowering of sodium and chloride in the serum which in the case of heat cramps is due to loss in the sweat without adequate replacement.

MacCance[72] emphasises the importance in salt loss/

loss of sodium, which is the chief constituent of the base of extracellular fluids, and Derrick[132] found ammonium chloride to be ineffective in relieving miner's cramp, which is an indication of the importance of the sodium element. The renal threshhold for sodium is apparently controlled by the suprarenal cortex, and in the absence of its secretion, as in Addison's disease, forced excretion of sodium takes place with consequent changes in the water balance of the body. The rise or fall of the sodium concentration of the plasma alters its electrolytic osmotic pressure, and the cells of solid tissues respond to this by varying their water content. The changes in plasma volume, blood volume, cell count, serum sodium chloride, and urea in Addison's disease are the same as those in severe sweating with ingestion of large amounts of water, and the cause is the same - loss of sodium[72]. Talbott[89] mentions a case in which the presence of early Addison's disease increased the subject's susceptibility to Heat cramps. Swingle[133] finds that adrenalectomised animals can be kept normal in health with very low serum sodium and chloride if adequate amounts of cortical hormone are given to ensure the mobilization and shift of tissue fluids to the extracellular spaces and blood stream. When more marked sodium chloride depletion produced by glucose injections has caused collapse, a single intravenous/

intravenous injection of 30% sodium chloride solution restores the animal to normal health and vigour (134). During salt deprivation the urinary excretion of sodium and chloride is very small and Banting and Gairns (135) noted that although the increase in urea and non-protein nitrogen in adrenalectomised dogs seemed to depend on the ability of the kidney to excrete, the chlorides did not, and could be kept at normal levels in the blood whether the kidney was excreting or not. Intact dogs depleted of sodium chloride remain healthy with very low levels of serum sodium and chloride, as enough hormone is elaborated to restore the altered fluid distribution to normal without change in the level of serum sodium and chloride. Injection of the hormone rapidly reduces the blood concentration and blood urea to normal. Langdon Brown (136) points out that severer forms of the condition ascribed to salt loss in human subjects have been described as "functional hypoadrenalism", and have been helped by cortical extract, although the giving of salt is more rational and more economical. Achlorhydria occurs in these cases and salt deprivation is known to lessen the formation of hydrochloric acid.

For the experimental study in human subjects of salt deficiency, uncomplicated by forced dehydration, MacCance (72) gave a salt free diet combined with sweating/

sweating, avoiding over concentration of sodium salts in the body fluids by replacing the water lost in the sweating. The symptoms and signs produced included an obvious change in the facies, the temporal hollows and cheeks falling in, and the eyes becoming tired and sunken. There was loss of weight, anorexia and nausea, and a curious sensation of loss of taste probably responsible for the apparent thirst complained of. Evidence of the abnormal water metabolism was the absence of diuresis after copious water drinking, the diuresis not developing until much later. The pulse volume was small, and the blood pressure normal. There was a sense of exhaustion, and a curious lassitude and apathy. Cramps were troublesome, and occurred in the chest after coughing, in the floor of the mouth after yawning, and in the fingers. The cramps were less severe than Heat cramps, probably because the desalting occurred gradually. The resemblance to Addison's disease was marked except for the pigmentation and the low blood pressure characteristic of that disease, which he suggests may be due to the absence of the medullary rather than of the cortical hormone. The loss of chlorides and sodium amounted to about 30% of the total body contents of these substances, and some came from the plasma, some probably from the cells of glandular organs such as the kidney, and brain, but the main source was extra/

extracellular fluids such as lymph and interstitial fluids, which must have been greatly reduced in volume. In the recovery period the loss of sodium was almost all regained, but the loss of chloride was more than made good. Sodium is the controlling ion, and during deprivation some sodium lactate and bicarbonate were lost as well as sodium chloride, but during recovery sodium chloride only was returned to the body which retained it. The changes in the water balance are due to the absence of sodium which is at first associated with loss of weight, anhydraemia, and haemoconcentration, the osmotic pressure being normal. The loss of weight, however, suddenly ceases as the body sacrifices its osmotic pressure to maintain the volumes of the plasma and **extracellular** fluids, and during this period when the osmotic pressure of the plasma is reduced the irregularities of the water regulation occur. The venous blood becomes viscous and a rise in cell count, haemoglobin, cell volumes, and plasma protein indicate anhydraemia. The colloidal osmotic pressure of the plasma is thus raised as it is in pure dehydration, but on account of the fall in serum sodium and chloride the electrolytic osmotic pressure is low. The excretion of urea is not normal and the blood urea is high, although the power to concentrate urea is normal. Possibly the cause is reduction of glomerular filtration, or there may/

may be reabsorption of urea to raise the subnormal osmotic pressure of the plasma. There is a negative mitrogen balance possibly due to autolysis of tissues resulting from malnutrition from the reduction in the volume of the blood and interstitial fluids, and the impaired circulation due to viscosity of the blood. Although sodium bicarbonate as well as chloride is lost the alkali reserve does not fall, and there is a tendency to alkalosis. It will be remembered that Maizels and Smith[77] found that the plasma bicarbonate varies inversely with the chloride. A general tendency to alkalosis has been observed in water intoxication also[129], and Oakley[137] found that a rise in alkali reserve and blood urea was caused by much smaller doses of alkali than usual in cases of severe or continued vomiting with loss of chlorides.

Maizels and MacArthur[138] found that when more salt was lost than water, bicarbonate is retained to restore the osmotic pressure of the plasma, and that when the osmotic pressure has been very low the retention of base may be so marked as to leave the urine acid.

The increased urinary ammonia after loss of sodium and potassium which might seem to indicate a tendency to acidosis was found by Atchley[87] not to depend on the pH of the blood. Pure sodium chloride depletion apart from dehydration appears therefore to cause/

cause a tendency to alkalosis. MacCance[72] studied the effect of the production of an intense temporary alkalosis by overbreathing, during salt depletion. The normal response to this includes diuresis with an alkaline urine due to increased excretion of sodium and potassium and sometimes of chloride as well.

During salt deficiency, however, there is no change of reaction and no increased excretion of base, for the kidney during salt deficiency is excreting practically no sodium, and it does not do so when the plasma becomes alkaline, as it must attend to the regulation of the osmotic pressure of the plasma rather than to that of its pH.

The excretion of potassium also is not increased, and this is because there is a general depression of renal activity. The possibility that the depression might be due to diversion of blood from the kidneys by the muscular action of overbreathing was excluded by the observation that there was no significant change in renal activity when overbreathing was performed with air mixed with carbon dioxide, thus doing the same muscular work but without the production of alkalosis. The kidney, then, is evidently seriously hampered by alkalosis combined with sodium deficiency.

There can be no doubt in view of these metabolic changes which have been described, that in addition to/

to the well known acute effect of salt loss in the causation of Heat cramps, the more gradual desalting of continued loss of sodium chloride in sweat without adequate replacement, must be considered as a possible cause of illhealth in hot climates.

. . . .

HYPERPYREXIA.

If the external heat is very great, or the body's capability for heat loss is inadequate, the body temperature may rise to dangerous heights before thermal equilibrium between the body and its environment can be established, and the condition of Heat Hyperpyrexia which is the typical dramatic form of Heatstroke, results. A temperature of 107°F or over is commonly regarded as constituting hyperpyrexia[83] and Lee[20] believes that the critical temperature is a little below 108°F, at which temperature neuroglobin separates out. Brief exposure to 109°F or more prolonged exposure to 107° or 108° result according to Wright[4] in death of the cells of the central nervous system and coagulation of their substance, and a similar figure is given by Pembrey.[2] Adami[139] stated that 107.5°F is the upper limit of temperature compatible with life but Evans[41] gives the higher figure of 111.2°F as that at which death ensues, and still higher temperatures have been recorded. Wunderlich's case of Tetanus with a temperature of 112.5°F was quoted by Coats[140] and by Hamilton[141] who believed such a temperature to be quite exceptional, and that any instances recorded of temperatures higher than this should be regarded with suspicion. The highest/

highest temperatures without death occurring which are recorded by Flack and Hill[142] Tigerstedt[143] and Noël Paton[144] are respectively 113°, 114.8°, and 115.8°F, and Schäfer[145] mentioning a case of Scarlet Fever in which the temperature was 114.2°F described a temperature of 113°F as extremely dangerous, and 116.6°F as quickly fatal to mammals.

A rectal temperature of 110°F is by no means uncommon in Heatstroke and Manson Bahr[71] states that the temperature may rise to 113°F for a short time and the patient may yet recover. MacCallum[146] mentions a case described by Lambert in which the rectal temperature reached 117.6°F, and a case of his own in which the temperature after death rose to nearly 120°F. The duration of such high temperatures has never been long[64] and accepting Schäfer's[145] opinion and that of Burton Opitz[147] that the body cannot long withstand a temperature of 111° to 113°F, it will readily be realised how grave are such cases, and how easily they may prove rapidly fatal if steps are not taken to lower the temperature.

The condition is predisposed to by anything which stimulates heat production, such as infections, alcohol, hyperthyroidism, and hard muscular work, or by anything which interferes with heat loss such as the wearing of unsuitable clothing, atmospheric conditions/

conditions which are not conducive to the evaporation of sweat, dehydration, and the inhibition of sweat. The influence of such factors in the causation of effects of heat will be discussed later. It is possible that the existence of one of them may be necessary for the production of Heat Hyperpyrexia, and of them all the inhibition of sweat might well be the most potent. Suppression of sweat is very commonly seen in cases of Heatstroke and many regard it as a most important etiological factor. Hearne[148] [149] believes that suppression of sweating, which may be present from one to forty eight hours before the attack, and may come on suddenly, is due to exhaustion of the sweat glands, and that the defect may be localised in the sweat glands themselves. This suppression of sweating he believes to be most important in the etiology of the acute type of Heat Hyperpyrexia with unconsciousness and possibly convulsions and muscular spasms, the lowest temperature required to produce this clinical picture being, he finds, 108°F, though temperatures of 109 or 110°F are more usual.[149] Dryness of the skin is common in the early stages of such cases[150] and Pike[151] found that suppression of sweating invariably preceded the occurrence of Heatstroke, whilst Howard[152] found that the encouragement of free sweating prevented the occurrence/

occurrence of Heatstroke amongst convalescents from various diseases. Love[153] found that the administration of atropin to patients in hot weather before an anaesthetic predisposed to Heatstroke, though it may be pointed out that the anaesthetic itself would render the heat regulating mechanism less active.[150] Ferris[154] noticed that, in a patient in whom spontaneous recovery occurred, the fall in body temperature coincided with the onset of free sweating, from which as well as from observations on other patients he concludes that absence of sweating may have some effect in causing Hyperpyrexia.

The "after fever" of Heatstroke, regarded by some as toxic in origin, Hearne[155] believes also to be due to suppression of sweating which persists for one or two weeks after the attack, during which time the sweat glands do not even respond to pilocarpine although it causes salivation. As already mentioned, Hill[55] attributed the occurrence of batches of cases of Heatstroke as the temperature rose in stages, to differences in men's ability to sweat, and Kuno[32] quotes Hiller's observation that Heatstroke developed more easily in soldiers who had been swimming, which was ascribed to depression of sweating by cold. In Kuno's own experience, however, the suppression of sweat occurred after many other symptoms of Heatstroke had/

had developed and could not therefore be regarded as the cause. Kuo and his coworkers[50] studying experimental Heatstroke found that sweating ceased long after many severe symptoms had occurred, and when the subject was about to fall into a stuporose condition. They believed, therefore, that suppression of sweat was not a factor in the causation of Heatstroke, nor was it due to increased body temperature, nor to exhaustion of the sweat glands, since it was rapidly restored on returning to cool air, and whilst the body temperature was still high. The fact that it was suppressed or restored concomitantly over the whole body surface suggests some inhibitory effect upon the sweating centres operative only when the centres are actively excited. It was the fact that sweating becomes progressively less copious with increase in duration, and that the percentage of sodium chloride rose with prolonged sweating which, as we have seen (pp.63,64), led Haldane[84] to believe that exhaustion of the sweat glands occurs. The influence of salt loss on sweating has already been discussed. Whilst certain degrees of salt loss are associated with excessive sweating, severe depletion may result in a depression of sweating (pp.67,70). Whatever is the mechanism of its production, suppression of sweating would have a profound effect upon the regulation of body temperature, and it seems probable/

probable that in certain cases it may play a part in the development of Heatstroke, and even when not a causal factor it is an indication of the gravity of the patient's condition.

When the thermal adjustment fails and an uncontrolled rise of temperature occurs, a vicious circle is established, for as the body temperature rises, the heat production independent of work also rises as a result of increased metabolism. Even the slight increase in body temperature occurring when the external temperature rose from 30° to 45°C was found by Grollmann[25] to cause an increase of 20 cc per minute in oxygen consumption. This he ascribed to the increased reaction velocity of chemical processes occurring in the cells, and to increased activity of the respiratory mechanism, and heart, and of the sweat glands which, Kuno[32] notes, produce at least 1/13th of the heat which they can eliminate from the body. The arterio-venous difference decreases from

60 what?

60 at room temperature to 46 at 45°C because of the great increase in blood flow through the skin where metabolic activity is relatively small compared to its blood supply. Metabolism as well as being increased may become disordered, and the products of abnormal metabolism have been suggested as a further cause of suppression of sweating, and as a contributory cause of/

of deaths occurring at a late stage after hyperthermia has subsided[2]. Indicanuria may occur and is taken by some to indicate a process of autointoxication[159] which Morton[160] attributes to the failure of the excretory organs to get rid of the products of metabolism. A prodromal period of malaise, weakness, constipation, and diminution of urine he ascribes to this autointoxication and suggests as a possibility the production of some histamine-like substance in the body as a result of faulty metabolism.

Sir William Willcox in a discussion on Dr Marsh's[64] paper at the Royal Society of Tropical Medicine emphasised the importance of autointoxication in the production of Heat Hyperpyrexia, but Professor Pembrey disagreed with the theory of toxaemia, and said he thought that the condition depended upon a disturbance of the salt balance.

In experimental heatstroke Marsh[64] found great increase of the blood lactic acid, decrease in the chlorides of the blood and cerebrospinal fluid, absence of chlorides from the urine, and a decrease in the plasma carbon dioxide. He observes that the essential phenomena of the condition are independent of salt concentration since they occur in the non-sweating rabbit. Although, however, rabbits do not lose salt by sweating, Heller and Smirk[161] found an increased/

increased extrarenal loss of water in association with the reduced urinary output of rats and rabbits due to the slight increase in body temperature at a temperature of 98.6°F, which might result in alterations of the salt concentration of the blood. Wakefield and Hall[162] in their studies of experimental Heatstroke found similar changes in blood lactic acid, and a reduction in the alkali reserve, but the blood chlorides were increased in most cases. Though nitrogen retention is not an essential part of the early acute condition they found that an increase in non-protein nitrogen may occur due to rapid destruction of protein on account of the high temperature. Morton[160] attributes the increase in blood urea which may occur some twenty four hours after the initial attack, to damage to the renal parenchyma. Wakefield and Hall[162] record the fact that in 1863 H.C. Wood noted an "acid reaction in the blood", and Lee[20] describes acidaemia, a rise in blood lactic acid and a fall in the plasma bicarbonate together with a rise in non-protein nitrogen, as "terminal events". Wakefield and Hall[162] found that ketone bodies were absent, and Manson Bahr[71] and Willcox[59] record the presence of acetone and diacetic acid in the urine in only 12% of cases, the latter stating that the reaction was only of moderate intensity and concluding that Heat Hyperpyrexia is not due/

due to an acid intoxication. Although in ketosis there is frequently no increase in the Hydrogen ion concentration of the blood it is probably the presence of acetone bodies in the urine in some of these cases which has led to the giving of intravenous sodium bicarbonate somewhat indiscriminately to such cases, and it is therefore important to realise that a tendency to alkalaemia may be present in some cases. The rapid breathing resulting from a rise of body temperature may be much accentuated by conditions interfering with heat loss, such as moist heat, and this over-breathing results in the washing out of carbon dioxide from the alveolar air, lowering the Carbon dioxide tension of the arterial blood (2) (3) (55). Bayliss[(163)] found that the blood at 100.4°F was just on the alkaline side of neutrality. An acid urine need not negative the existence of alkalosis, especially in cases where there has been excessive salt loss, since as we have seen in this condition, the loss of bicarbonate in the urine may be avoided in order to prevent lowering of the plasma osmotic pressure (2) (73) (77). Lee and Mulder[(65)] found that the respiratory changes in response to high temperatures are slight for the first three hours, and when later they become more marked the increase is in depth rather than in rate, and the alveolar carbon dioxide tension does/

does not fall until the respiratory volume has been markedly increased. They found no conclusive evidence of a true alkalaemia of arterial blood. It may be concluded that the changes in the acid base balance are not of a constant nature, and that the variations in it probably depend not only on disordered metabolism, but upon the addition to the picture of salt loss or dehydration each of which as already described, has considerable influence upon the acid-base balance.

Nervous symptoms are prominent in Heat Hyperpyrexia, and the damage done to the central nervous system may lead to a fatal result, or, in the event of recovery, to various after effects. Partial coagulation of the proteins of voluntary and cardiac muscle from the high temperature may also occur, and assist in the production of cardiac and respiratory failure[(2)]. The myosin of heart muscle is said to coagulate at 113°F. Embarrassment of the circulation must result too from the presence of the great volume of blood in the skin, lessening the amount available for more vital parts of the circulation, whilst alkalosis if present may cause a fall in cardiac output and blood pressure from imperfect relaxation in diastole. Stitt[(164)] believes that the immediate cause of death is tetany of the heart muscle resulting from the accumulation of acid products due to the profound metabolic changes following on the breakdown of the heat regulating mechanism/

mechanism, and in 1917 Mayer[165] suggested that death from high temperature was due to the accumulation of acid in the tissues.

MORBID ANATOMY.

The body temperature remains high after death, and may even rise to as much as 120^{o}F, [146]. This may have been the cause of the continued warmth of the corpse of a Portuguese lady, which, six hours after death, was as warm "as in a living case of fever", which caused the explorer Livingstone to delay her burial until unmistakeable signs of putrefaction occurred.[166]

Rigor mortis is unusually rapid in appearance, and putrefactive changes commonly occur early. Congestion of the dependent parts of the body may be marked and petecheal patches in the skin, mucous membranes, brain, medulla oblongata, and viscera are seen in some cases. (150) (159) (167). The blood is dark, unusually fluid, or only partially clotted, and may be less alkaline in reaction than usual; the red blood corpuscles have been said to be crenated and not to form rouleaux, and some lysis of polymorphs may be seen. (71) (150) (164) (168) (169). The brain and meninges are congested and sometimes oedematous, and there may be an increased amount of serum below the pia/

FACTORS PREDISPOSING TO EFFECTS OF HEAT.

ATMOSPHERIC CONDITIONS.

Before the clinical conditions due to heat are described, the factors which predispose to these conditions, and the general effects of hot climates on white men will be considered.

Of the environmental factors which influence the regulation of body temperature the atmospheric conditions are the most important, and a brief description of the chief factors governing atmospheric conditions and of some of the instruments and methods of calculation used in this connection is contained in Appendix II.

The body is able to withstand exceedingly high temperatures if the air is dry, as heat loss by evaporation of water is often able to balance that gained by radiation and convection, as well as that produced in metabolism. Payne[171] stated that in dry air, temperatures of 210°F or even 260°F have been borne for several minutes, and that even higher figures have been given. Work may be carried on for some hours, at from 120° to 160°F, and in the Tropics a dry temperature of 120° to 130°F is sustained for a long time without damage and with only a slight rise of temperature. Blagden, Fordyce, and Dobson,[(173) to (174)]

were/

were able to remain in a room in a dry heat of from 210° to $260^{\circ}F$ for some time without serious inconvenience and with only moderate tachycardia and slight rise of temperature, although beefsteaks when exposed in the room were cooked in thirteen minutes.

The presence of wind, however, alters the state of affairs, and a hot dry wind may bring about dehydration quite rapidly, as occurred recently in the case of the forced landing of the aeroplane "Horsa" in the Arabian desert when the air temperature was 125° to $130^{\circ}F$. Stott[175] describes how "the scorching breeze like a blast from a furnace sucked fluid through the skin sweat glands, which immediately evaporated so that no visible sweat was produced. This evaporation, no doubt, caused some slight cooling of the skin surface, which made us choose to lie where the breeze was. But such temporary cooling was produced only at the vital expense of the invisible constant drain of body fluids through the skin to the atmosphere". According to Hill[176], with an air temperature over $98.5^{\circ}F$, the effect of wind is to increase the body heat if the skin is dry, and a hot dry wind by drying the skin more quickly than it can be kept wet by sweat directly heats the blood, and prevents a certain amount of reflection of the sun's rays which occurs when the skin is wet with sweat. (55) (70) (177).

The/

The type of Heatstroke due to these conditions Mouriquand[178] calls "Coup de chaleur seche", and observes that it is encountered on hot days in continental climates, in the Sahara, and in centrally heated rooms. Apart from such conditions, however, dry heat can be much better borne than moist heat, and Mouriquand[178] considers "Coup de chaleur humide", the type met when the humidity is high, to be the characteristic tropical Heatstroke, and says it is also the type encountered in mines, near boilers, in kitchens and on washing days. The influence of humidity on temperature regulation has been proved by many investigations which need not be detailed, and the importance of high humidity in the causation of Heatstroke is universally recognised (2) to (4) (11) (18) (36) (41) (65) (150) (177) to (189).

Complete saturation of the air with water vapour when the temperature is 90°F has been found to lead to an uncontrollable rise of temperature,[(64) (187)] and when the external temperature is above 98.6°F, air movement is unable to counteract the effect of the high humidity[188].

Non sweating animals, too, have difficulty in regulating their temperature in moist air, and Lozinsky[190] found that temperature regulation in the dog fails and the blood becomes concentrated if the temperature/

temperature is over 91.4°F in moist air, but only if it is over 107.6°F in dry air. When the air is still the body is surrounded by a layer of air with a temperature intermediate between that of the body and that of the atmosphere which prevents heat loss (2). Whilst this makes a low temperature more easy to bear, it makes a high temperature much less endurable. Loss of heat is greatly assisted by brisk air movement which replaces the stagnant layer of air round the body and increases the amount of air brought into contact with the skin, and so utilizes the heat capacity and conductivity of the air to the fullest extent. Brisk air movement is especially important as an aid to heat loss when the humidity is high, as it increases heat loss by conduction and convection, as well as by evaporation when that is possible (3) (11) (55) (70) (150) (177) (191) (192).

The influence of the three important factors, Temperature, Humidity, and Air Movement, on temperature regulation may be summarised as follows. Increased air temperature lessens heat loss by radiation, conduction, and convection, and more must therefore be lost by evaporation. Heat loss by evaporation is checked by a high relative humidity but aided by air movement replacing the layer of hot moist air round the body, and stimulating the skin capillaries. If the temperature is high and the relative humidity low there/

there may be excessive loss of water with resulting thirst and dryness of the skin and mucous membranes, or even dangerous dehydration, which is especially liable to occur if there is much air movement. In cold weather a high relative humidity by increasing the conductivity of the air causes excessive heat loss, whilst too much air movement causes chilling.

These physical conditions are generally recognised as being of greater importance in ventilation than are chemical conditions such as the amount of carbon dioxide, or bacteriological conditions. Deaths in such circumstances as in the overcrowded airless holds of slave ships, or in the Black Hole of Calcutta were essentially deaths from Heatstroke, [70] [177] although excess of carbon dioxide may also play a part in such conditions[178].

In badly ventilated factories or mines it is chiefly the imperfect elimination of heat which causes illhealth and reduces the efficiency of the workers. The subject has consequently been extensively studied in order to determine the ideal atmospheric standards for health and efficiency. Relative humidity, and Dew Point (see Appendix II) and the Wet bulb temperature have, on account of the importance of moisture of the air, been adopted as indicators of comfort. Thus it has been found that the limits for comfort are/

are when the relative humidity is between 72 and 77%, although lower figures have been found preferable in America possibly because in that country higher temperatures are usual[193]. If the relative humidity is over 85% and the temperature is also high, say 90°F or over, the danger of Heatstroke is considerable. Hill[176] cites J.L. Bruce as taking the Dewpoint as an indicator of comfort. Haldane[191] held that in still warm air the all important factor is not the air temperature, nor yet the absolute percentage of aqueous vapour present, but the temperature shown by the wet bulb thermometer. If this exceeds 78°F, continuous hard work becomes impossible, whilst above 88°F it becomes impracticable for ordinary people to stay for long periods in such air. In moving air the limit is extended upwards by several degrees. So long as the wet bulb temperature is low enough, evaporation can take place, and cooling of the body can be maintained despite a very high dry-bulb temperature. Recently Bedford[194] has found that the dry-bulb air temperature is about as good an index of skin temperature as any other measure in use. The importance of air movement, however, which was recognised by Heberden[195] in 1826, led to the realisation that estimations of temperature, humidity, or vapour pressure were of less value than an estimation of the cooling power of the air, which takes air movement also into consideration. Such/

Such an estimation can be made with the Katathermometer (described in Appendix II) after the introduction of which Hill[196] was able to claim that exclusive study of the wet bulb temperature, or the dewpoint, and the dogma that the relative humidity must be kept at 75%, must yield to estimation of the cooling power or degree of comfort of the atmosphere. Readings with the dry-Katathermometer give the heat lost by radiation, conduction, and convection, and with the wet Katathermometer that lost by evaporation is also shown, and the difference between the two gives the cooling power due to evaporation. Air velocity can also be measured with the instrument by the use of the Nomogram (reproduced in Appendix II) devised for estimating cooling power and air velocity from the Katathermometer readings[197], a special high-temperature Katathermometer being used when the temperature is over 90°F.[198] Charts and formulae for use with the instrument have been worked out by Yaglou[199] and by Weeks[200] [201] who has also devised an instrument, the Coolometer which fulfils the same functions as the Katathermometer. Other instruments used for estimating comfort conditions are the Air Tester of Menzies and Frederick[202] and Yaglou's Heated bulb thermometer[203].

With the Katathermometer important observations have been made upon the physiological aspects of climatic conditions, of ventilation in industry and of the/

the influence of clothing (176) (177) (204) to (208).

Estimations of the cooling power of the air and the air velocity, combined with readings of the dry and wet bulb thermometers give an indication of what improvements in the atmospheric conditions are possible, and how they should be attempted. Various standards have been worked out, and it may be said that an indoor dry reading of 6 or wet reading of 18 indicate an invigorating atmosphere. A difference between the dry and wet readings of 12 indicates a comfortable atmosphere, whilst if it is less than 12 the atmosphere tends to become hot and stuffy, and if it is more it may be too cool for ordinary work. It should be remembered as Hill[(70)] points out, that, whilst it affords an excellent guide to the cooling power of the atmosphere to which the human body, dry or sweating, is exposed, it does not actually measure the rate at which heat is lost from the human body. It measures the rate at which heat is lost from its own surface, which in volume and surface is comparable only to the end part of the human thumb. Yaglou[(209) to (211)] holds that this difference between the thermal properties of the Katathermometer and those of the human body is so great that, although an excellent anemometer, it does not fulfil the purpose for which it was devised, and is a less sensitive index than the so-/

so-called "effective temperature" scale. This scale depends upon the fact that for a given degree of warmth or comfort, temperature, humidity, and air movement may vary considerably, so long as their combined effect remains the same. Different combinations of the three factors give what are called equivalent conditions. A scale of equivalent conditions has been worked out and this scale not only indicates the warmth felt by the body, but also determines the physiological effect produced on it, and hence is known as the "effective temperature scale"(209). Physiological reactions were found to follow this scale very closely, regardless of wet bulb, dry bulb, dewpoint, relative humidity, or air movement. Haldane's view(191) that the wet-bulb temperature is all important, and the dry-bulb temperature of no significance, is challenged by exponents of the effective temperature index who maintain that, even at high atmospheric temperatures, the dry bulb temperature has considerable influence. Thus whilst Haldane, to secure comfort conditions in mines would aim at reducing the humidity, Yaglou(209) recommends increasing the humidity so as to bring the dry-bulb temperature nearer to the wet-bulb temperature, and so to lower the effective temperature. He points out that whilst the use of air movement is the simplest and most inexpensive way of cooling the air, the benefit derived therefrom at/

at very high temperatures is small, and the effective temperature must be lowered by other means before the air is set in motion. This can be done by the evaporation of water, since when unsaturated air is brought into contact with water a certain amount of heat is abstracted from the air and its temperature is considerably lowered. Unless heat is added to or subtracted from the system the wet-bulb temperature remains the same, and the dry-bulb temperature increases until it becomes identical with the wet bulb temperature, saturation point being reached. When this saturated air is set in motion its cooling power is very great, and acting on this principle the use of humidifiers and blowers was found very useful for conditioning plants where the temperature was high and the humidity low. The influence of the dry bulb temperature on effective temperature is illustrated by a table in Appendix II, page 249. The effective temperature is determined by charts from the dry and wet bulb temperatures, and the air movement, and various standards of comfort have been worked out. For example, at rest, the comfort conditions are between 63° and 71°F on the effective temperature scale, and in summer between 64° and 79°, whilst the upper limit of temperature efficiently endured with muscular work is 80° E.T. (209) (212) (213).

Clothing/

Clothing markedly affects the comfort zone and at rest stripped to the waist comfort conditions are between 66° and 82°, the optimum being about 72.5°F.[(214)] Some of the conclusions which Yaglou based upon the effective temperature scale were challenged by Vernon and others[(215) to (218)] who held that the Katathermometer afforded a better index of comfort conditions, and that it was an erroneous principle to increase the relative humidity, and to lower the dry bulb temperature, whilst leaving the wet bulb temperature unchanged.

It was also pointed out that American workers seem to prefer a somewhat higher temperature than do British workers[(219)]. More recently, however, Vernon[(220) (221)] has found himself more in agreement with the supporters of the effective temperature scale, and believes that it may be possible to obtain a single index to represent the physiological responses of the body to the combined effect of dry and wet bulb temperature, air movement, and radiation, but points out that acclimatisation must always be taken into account. Without some such single index the opinions of observers in different parts of the world, as to the critical temperature at which Heatstroke is liable to occur, are bound to vary. Marsh[(64)] in the Persian Gulf found that the frog suffered from Heatstroke when/

when the shade temperature was over 104°F, the guinea pig at 100°F, the rabbit at 116°F and man at about 130°F. Willcox[159] found the danger point in Mesopotamia to be 110°F, and 120°F or over to be very dangerous; Hutchinson[222] in Allahabad put it at a maximum temperature of 110°F, with a minimum temperature of over 85°F, and a mean temperature of over 98°F, whilst Morton[160] considered that in Iraq the critical temperature was from 119° to 120°F, and that the factor of humidity did not enter into the question. Willcox[159] points out that the effect of heat is cumulative, and that a heat wave, or succession of hot days is of great importance, a fact emphasized also by Rogers[181] by Fremantle[223], and by Hutchinson[222] who found that a period of hot dry weather, especially if the nights were hot, produced most cases although he thought that a high wet-bulb temperature might be associated with the occurrence of severe cases. Haldane's views[191] on the importance of the wet bulb temperature are reflected in the observations of Pembrey[224] who found that of fifty cases of Heatstroke amongst soldiers in India, twenty five occurred when the wet bulb temperature was 80°F and over, and eight when it was 84°F and over. Hamilton[225] states that no cases occurred in Allahabad when the wet bulb temperature was below 75°F and the dry bulb temperature below 88°F, and Martin[21] found that the maximal wet bulb/

bulb temperature endured in Palestine was 87°F, and that it then made no difference if the dry bulb temperature was 86° or 100°F. These estimations of the danger point do not take into consideration the interplay of Temperature, Humidity, and Air Movement, and it is possible also that local differences may be produced by other factors which Mouriquand (178) believes may play a part, such as barometric pressure, electricity, and various elements comprised in the term "indéterminé météorologique". He finds that some individuals whom he classes as "météorolabiles" are more susceptible to atmospheric conditions, and Llewellyn, (226) who considers Rheumatism to be essentially the outcome of inadequacy or instability of the heat-regulating mechanism, observes that rheumatic subjects are abnormally sensitive to changes in humidity, temperature, and barometric pressure. Observations with the Katathermometer or use of the effective temperature index would yield more definite information as to the climatic conditions leading to prevalence of Heatstroke, and by a series of observations the danger points in various districts could be determined so that medical officers could know when it was especially important to enforce prophylactic measures, whilst the readings would also afford an indication of the form which some of these measures should take. Whilst extremely accurate observations may not in military practice/

practice be so essential as they are in industries in which modern air conditioning is in use, it is nevertheless possible to do a certain amount to improve atmospheric conditions in Barracks and Hospitals, on the lines indicated by such observations, whilst Katathermometer readings can afford a guide as to whether a certain ward or bed is a suitable one for the treatment of Heatstroke cases.

SOLAR RADIATION.

The part played by the sun in the production of the conditions described as 'Effects of Heat' has for many years been a much debated question.

The term "Sunstroke" is firmly fixed in popular usage and also in medical nomenclature, but opinions are divided as to whether it should be regarded as a separate entity. Some writers describe Sunstroke as a distinct clinical condition, attributed usually to the action of the sun's rays on the Central nervous system[(227) to (234)]. Castellani and Chalmers[(169)] reviewing in 1919 the opinions of many authorities found that it was generally recognised that the action of heat and the action of sun's rays were separate, but they described both Heatstroke and Sunstroke as one clinical condition under the name of Thermic Fever which was used by Wood[(235)] who did not believe in any special action of the sun's rays and who disliked the term/

term Sunstroke. Manson Bahr[71] observing that there is some special element in the solar rays capable of injuriously affecting the tissues particularly if they have not become gradually habituated to sun exposure, describes in addition to Heat Exhaustion and Heatstroke, the condition of Sun Traumatism. Corbusier[228] described two forms of Sunstroke, Siriasis or Insolation, due to the actinic rays of the sun, and Sunstroke or Sun Traumatism due chiefly to heat, but in which, he thought, the actinic rays might also play a part. Belief in the pathogenicity of the actinic rays led to the recommending of specially woven coloured fabrics designed to afford protection against these rays, and of yellow or red linings for sun helmets[236] to [239]. Extensive tests with orange red clothing carried out by Phalen[240] proved that protection from the actinic rays of the sun, afforded no immunity from the physiological effects of the climate of the Phillipines, which he concluded were due to moist heat without the aid of the sun's rays. The wearing of orange red clothing, in fact, increased the body heat and caused excessive perspiration. As regards the coloured linings in topis, Blackham[241] found that the temperature inside such helmets was actually higher than in other forms of helmet, and Hill[177] concluded that colour is immaterial and that proper ventilation of the topi is all important. The belief expressed by/

by Joyeux[230] that exposure of the head to the sun for a few minutes in the heat of the day is enough to cause Sunstroke is very widespread amongst the laity, so that as Megaw[150] points out, many carry their dread of the sun to dangerous limits, remaining indoors in darkened rooms all day, and losing the healthful stimulation caused by the Ultra-violet rays. Megaw believes that, although all the rays of the sun tend to be more powerful in the Tropics than in cool climates, they have no mysterious dangerous properties such as are often attributed to them, and the real harm done by them consists in their powerful heating effect on the body, adding to the risk of Heatstroke in trying atmospheric conditions. This view that "Sunstroke" is merely a form of Heatstroke and not due to any specific action of any rays of the sun other than the heat rays, all the symptoms attributed to "Sunstroke" being capable of being produced by heat without any exposure to the sun, is very widely held (2) (18) (20) (89) (159) (160) (164) (242) to (252). Willcox[252] considers that "it has not been shown that any rays of the sun other than the heat rays have a special significance in the causation of Heatstroke" but "since the temperature in the sun greatly exceeds that in the shade exposure to the sun's rays is a very important exciting cause". In Lee's[20] opinion "bright/

"bright tropical sun falling vertically on the head and neck may, where the mean body temperature is just subcritical, serve by a local heating effect to raise the temperature of the Central nervous system to the critical point, but apart from this can have little topographical effect on the causation of hyperpyrexia". The same view was held by Hill (55) (70) (176) (247) who in 1920 found that the evidence was conclusive that "Sunstroke" is due purely to overheating of the body, and pointed out that the heat of the sun in the desert for example, greatly affects the heat of the body, and that even in the shade radiant heat from sand and skyshine must be considerable. He confirmed Sonne's observation that the luminous rays heat up the subcutaneous tissues to a greater extent than do the dark infra-red rays, and found that exposure of rabbits' bodies to sun could cause a considerable rise of temperature in all deep tissues. Although Bushnell[(253)] observed that infra-red and heat rays have little penetration, Hill[(254) (255)] found that, although no short ultra-violet radiation penetrates below the surface of the derma, much of the long ultra-violet, the visible and the short infra red rays penetrate to the blood vessels in the derma and the red rays penetrate the blood circulating in those vessels. Barbour[(157)] found the heat conductivity of the cranium to be relatively good, and MacLeod[(256) (257)] noted/

noted that heat can penetrate deeply even into relatively vascular tissues, and when locally applied can penetrate as deep as over 20 millimetres. He found that heat penetrates from the surface into the brain much more than it does in the case of the liver and kidneys, but less so than into muscle, and that the changes in temperature are more quickly produced.[169] Discussing the experiments of Castellani and Chalmers in which rabbits exposed to the sun with their heads shaved were protected by coloured glass, Hill[177] points out that the coloured glass probably saved them from Heatstroke as it absorbs the heat as well as the light rays. The heat into which the energy of the rays which penetrate and are absorbed is converted, is carried away and distributed by the circulation so that with an efficient heat regulating mechanism no harm should result. Marsh's[64] experiments proved that the sun's rays have no direct action on the brain tissues of exposed rabbits as it was impossible to injure them by exposing their heads to the sun provided their bodies were kept cool. After prolonged exposure of the shaven heads of rabbits, whilst their bodies were kept cool, he found no changes in the nerve cells of the brain and no capillary thrombosis. Exposure of their bodies to the sun whilst the heads were kept cool killed the rabbits after half an hour's exposure. He maintained therefore that heat alone is responsible/

responsible for "Sunstroke". Discussing his views Professor Castellani and Sir Charles Martin agreed that ultra-violet rays played no part in the production of "Sunstroke" but the latter said that he did not feel sure that "Sunstroke" might not exist apart from Heatstroke. Doctor Eidinow said that in experiments with Sir Leonard Hill[258] [259] he had found that animals could be killed by light alone apart from any heating effect, and in such animals he has found thrombi, areas of necrosis, stasis of the circulation, and intravascular clotting[260]. He believed that in such cases death might be due to the absorption of toxic substances produced by intensive ultra violet radiation, or by irradiation with visible rays in the presence of photodynamic sensitization, and suggested the production of a histamine-like substance which causes a marked fall in blood pressure and failure of the circulation. In 1928 Eidinow[261] said that excessive exposure to infra-red and visible rays causes Heatstroke, Lightstroke, and many of the dangers which menace the adaptation of white men to the Tropics, whilst in 1905 Woodruff[262] attributed to Sunlight many of the ill effects of tropical climates on white men, including affections of the Nervous system and even insanity. Believing that black bodies radiate heat to cooler ones more quickly than do bright bodies Woodruff held that inability to radiate heat causes the/

the white man to suffer from overheating in the Tropics as his temperature rises with that of the air. This Eijkman[263] calls "physical heresy", since the colour of a body has no influence on the emission of dark heat rays. Koch and Reed[264] found that pigment increases the radiation of heat, but Eijkman,[263] by noting the cooling rates of skin-covered boxes filled with water, proved the radiating power of white and brown skin to be the same and Hill[177] and Martin[21] found that white skin reflects more than black. Pigment is formed not as a defence against heat but for protection of the skin against the inflammatory action of sunlight[249]. This protection it affords by absorbing the light rays and transforming their energy into heat,[177] [265] and Eijkman[263] showed that a coloured skin absorbs more heat than a white one. The tax put upon the heat-regulating mechanism in getting rid of the heat into which pigment transforms the light rays is offset by the fact that the pigmented man can afford to have a thin horny layer, and dilated blood vessels for heat loss without risk from ground glare and sky shine, and can be naked and so secure the full advantage of heat loss by radiation convection and evaporation, whilst black skin around the eyes diminishes diffusive reflection into the eyes and so lessens sun glare, a protection which is often given to Indian children by artificial blackening/

blackening around the orbit. Although, then, some of Woodruff's views may have been based on a miscon-
p112
ception, Eidinow's observations, mentioned above, make it impossible to dismiss the action of tropical light as unimportant and its possible effects must be further considered.

Laurens[249], defining light as "that agent or force in nature by the operation of which on the organs of sight things are rendered luminous or visible", points out that the terms ultra-violet and infra-red "light" are misnomers as these rays do not render objects visible, and are therefore radiant invisible energy. Solar radiation as it reaches the earth's surface extends from 0.29 μ to 5 μ., though there is very little energy longer than 2 μ and it is often described as extending from 0.3 to 3 μ. On entering the earth's atmosphere 5% of the total radiation is ultra-violet, 52% visible, and 43% infra-red, but - owing to extraction and absorption by the time it reaches the earth's surface the distribution is ultra-violet 1%, visible 40%, and infra-red 59%. Only 75% of the total solar radiation reaches a level of 1800 metres and only 50% to sea level, so that the total intensity is greater at high altitudes. The amount of dust and humidity markedly affect the extent and intensity of the spectrum, and it has been found that radiation intensity in the Tropics differs little from that/

that in temperate zones, and in many coastal towns in the British and Dutch Indies with high humidity and much dust the maximum intensity is actually less than that experienced in June at mid-day on the North Sea coast. The proportions of the different rays too may differ in different places. Johnson[266] believes that in South India the proportion of infra-red rays is high, and Kennedy[267] observing that little is known about ultra-violet radiation in the Tropics, suggests that the physician in the Tropics could make valuable additions to knowledge by making occasional measurements of direct solar and diffuse sky radiation. He mentions several methods of making these measurements and does not advise the methylene blue method which is widely used[268]. According to Laurens[249] the great difference is in total radiation which shows large daily and annual variations. The radiation received at a given spot can be measured by Vernon's globe thermometer[269] which indicates the combined effects of radiation and convection as they affect the human body, the excess of the globe-temperature over the air-temperature indicating the "effective radiation temperature".

Although as far as heat rays are concerned, the amount of radiation in a unit of time may be little greater than in temperate climates, other climatic conditions, temperature and humidity, may allow of less/

less loss of heat, and Hill and Eidinow[270] found that the lethal action of light in infusoria may be increased by raising the temperature. Glare from sand and rocks has also to be considered. The action of solar radiation is summarised in the following table by Megaw[150] who points out that it is not absolutely accurate since blue and violet rays play a part in illumination, though to a less extent than the luminous rays, which in their turn produce heat though less than do the red and the infra-red rays.

TABLE.

PROPERTIES OF THE RAYS OF THE SUN. (MEGAW).

	Visibility.	Chief Actions.	Diseases Caused.
Heat Rays.			
1. Infra Red	Invisible	Heating	Heat Exhaustion
2. Red	Visible		Heat Fever
Luminous Rays			
3. Orange	Visible	Chiefly illuminating	Glare headache Night blindness
4. Yellow			
5. Green			
Chemical Rays			
6. Blue	Visible	Stimulating and anti-rachitic in moderate doses. Irritant and destructive of tissues in excessive doses.	Solar Dermatitis Pigmentation.
7. Indigo			
8. Violet			
9. Ultra Violet	Invisible		

French writers describe the three conditions of Sunstroke and Heatstroke - general and local effects due to heat rays - and Lightstroke - general and local effects due to the actinic rays, conditions which as Aimes[271] says are often difficult to separate and differentiate from one another. This triad is as Jausion[272] points out, really a "biad" (if one may coin this word in translation of "diptyque"), for solar radiation is a sum of heat and actinic properties, and in accordance with latitude, season, climate, and altitude, these two portions of the visible spectrum flanked by their invisible margins, ultra violet and infra red, neutralise one another. There is, he finds, an antagonism between the two ends of the spectrum, possibly a physical interference or a physico-chemical neutralisation of the effects of the ultra violet by the infra red. There may too be a physiological antagonism between the two halves of the spectrum, a fact supported by Hill[273] who does not believe that there is any physical antagonism. Thus vasodilatation produced by heat radiation carries away the toxins and tissue waste products resulting from actinic radiation, and the bactericidal action of short waves is partially checked by simultaneous exposure to the long waves of the spectrum. The French use of the term Sunstroke for solar dermatitis is a reason advanced by Megaw[150] for its abandonment in describing the type of acute Heat Hyperpyrexia which is often given this/

this name by English writers. It must be realised, however that in addition to the local effects of primary erythema from heating of the superficial skin layers by heat rays, of secondary erythema due to ultra-violet radiation and of haemorrhagic stippling, there are pronounced general effects resembling in some cases the clinical picture of Heat Exhaustion or Heat Hyperpyrexia. Thus in mild forms there may be general malaise, lassitude, vertigo and nausea. Grave forms with blisters and burns may have some elevation of the temperature, gastro-intestinal upsets, praecordial pain, and dyspnoea with congested facies and profuse sweating. When the burns are extensive many general symptoms occur from absorption of toxic substances, and suppression of sweat. There may be fever, oliguria, albuminuria, and signs of a definite toxic nephritis. The pulse is rapid, the temperature rises to 102° or $104^{\circ}F$ or more, there may be delirium, and sometimes an aseptic meningeal reaction as in Heatstroke.[271] [274] Lightstroke which Jausion[272] believes is rarely seen in man, occurs usually when the sun's rays are reflected, as on glaciers or at the seaside, and may occur in cold weather. Fifteen to twenty minutes exposure to the sun at a temperature of about 86° to $95^{\circ}F$ may be followed by primary erythema, succeeded later by secondary erythema, desquamation, and pigmentation, whilst by way of a general/

general reaction there may be malaise, lassitude, vertigo, nausea, headache and fever. Such cases point to the possibility that the actinic rays may have pathogenic effects apart from their action on the skin, and Jausion observing that d'Halluin and Lumiere ascribe to heat rays the important part in the deaths of guinea pigs exposed to the sun, himself believes that the actinic rays have some importance. Dr John Kinnear tells me that he is quite convinced that the general effects of the sun's actinic rays differ from those of the heat rays, and points out that with small doses of artificial sunlight mild degrees of general malaise and lassitude, as evinced by "crossness", are very easily produced in children, without the production even of erythema. Excessive exposure to light rays may, as Smith[275] says, entail not only fatigue and exhaustion, but may produce early degeneration of the skin, especially over the external ears, the lower half of the face, and the back of the hands, where the skin is atrophied, and pigmented, sometimes with warty growths or more serious local lesions such as Xeroderma pigmentosum or Epithelioma.[272] Bray[276] recently described a case in which angioendothelioma developed in a child after trauma superadded to sunburn. The effects on the skin of "photo traumatisme" whether due to heat or actinic rays, can be added to by what French writers call "photodynamisme", causing/

causing diverse effects according to various sensitizing agents exogenous or endogenous. They include among these conditions Pellagra the most typical of the "psychodermatoses", the connection of which with the action of actinic rays is hinted at by the occurrence of slight psychic changes accompanying the mild epidermal reactions produced by the therapeutic use of these rays.[272] The disturbances due to light are not confined to the skin, and ultra-violet light can excite or exalt the action of organisms or viruses, and cause eruptions of boils, acne or impetigo, for which the effect on the skin paves the way, a phenomenon called "Photobiotropisme". The most penetrating luminous rays acting upon the blood-forming organs, the granulocytes and macrophages, lower the threshhold of resistance to toxins, and thus predispose to conditions due to sensitization to various antigens, solar urticarias, prurigo or eczemas. This Jausion refers to as "photoanaphylaxis".

It is evident then that in addition to its local effects on the skin, and apart from toxic absorption from skin lesions, sunlight may cause various general effects. Experience with sunlight treatment of Tuberculosis makes it clear that it is unwise to risk such general effects when there is active Tuberculosis or when the patient is febrile.[277] Reactions to sunlight vary with individuals, and severe reactions may/

may be due to an abnormal sensitivity or to the presence of some light sensitizing substance.[278] Sufferers from Congenital Haematoporphyrinuria are excessively sensitive to light, as is shown by their unusual tendency to develop Hydroa aestivale.[66] [279] to [281].

Pigment is, as we have seen, a protection against the harmful effect of sunlight, and it has been observed that children who pigment well do better and put on more weight during sunlight treatment.[282] Those who are to be exposed to much sunlight in the Tropics or when climbing or skiing on high Alpine glaciers should take steps to acquire a good tan, and those who do not pigment well should adopt special precautions.

King Brown[283] mentions numerous suggestions which have been made to account for the physiological action of light. These include changes in temperature, changes in the physical properties of proteins, electro-chemical or electrical changes, such as changes in the electric potential of the red blood curpuscles, (284) (285), changes in surface tension, alterations in the metabolism of tissue cells, changes in capillary circulation, production of histamine-like substances,[260] and vibration effects.[286]

Clark[287] [288] believed that the effects were due to photochemical changes produced when light is absorbed,/

absorbed, and King Brown[283] mentions changes in the inorganic salts of the tissues and blood such as calcium and phosphorus. The activation of provitamin or the formation of vitamin may be an important factor. Reed[289] [290] observed that there seems to be some part of the ocular mechanism which is responsive to intense light, and that the responses affect the vaso-motor mechanism extensively, the predominant effect being depression. Pronounced depression of arterial blood pressure was produced when a beam of light was directed into one or both eyes of an anaesthetised dog, and also when the blood was irradiated. Leucopenia and lymphopenia, but relative lymphocytosis also resulted. In this connection it may be remarked that Tatchell[291] recently expressed the opinion that, although most cases are due to heat, Sunstroke does occur and is he thinks usually caused through the eyes. He believes it to be most common at sunrise and sunset, when the altitude of the sun is low, when as Kennedy[267] points out, the ultra-violet part of sky radiation considerably exceeds that of the direct sun. Perhaps the most interesting theory is that of Llewellyn and Basset Jones[292], who observe that the amino-acids tyrosine and cystine are stored in the skin, tyrosine being the mother substance of which the temperature-regulating hormones, thyroxine and adrenaline as well as melanin are chemical derivatives. Tyrosine enters also into the formation of insulin, another heat producing/

producing hormone, and the sulphur content of insulin is derived from cystine. Tyrosine and cystine exist in a free state in the blood, and have been detected in sweat, and the blood content of tyrosine and hence potentially of thyroxine and adrenaline is increased by ultra-violet radiation.

Cramer's views on the importance in heat-regulation of the thyroid adrenal mechanism have already been cited (pp.11,12) and this theory suggests an aetiological connection between sunlight and the hyperpyrexial type of case often described as "Sunstroke".

It would appear, therefore, that although in most cases heat is the most important factor, it is impossible to dismiss altogether the possibility that rays other than the heat rays of the sun may play a part in the production of the type of case known as Sunstroke or Heatstroke.

Various other factors, though of less importance than climatic conditions, may play a part in predisposing to Effects of Heat and these will be briefly considered here whilst the measures necessary to ensure that they do not adversely affect the health of the soldier will be described when dealing with the prevention of the Effects of Heat.

HOUSING.

A high incidence of illness due to heat may be expected/

expected amongst people living in unsuitable houses, in which for one reason or another the desirable comfort standards of air temperature, movement, and humidity cannot be secured, or in which there is overcrowding. No British soldier is likely to have to live under such conditions, for although some of the barracks abroad are old, all have been modernised to some extent, all are provided with electric fans, and are so constructed that comfortable standards are attainable. In peace time, tents and bivouacs are occupied only during the cooler part of the year, but in the event of war their use during severe heat might be necessary and special precautions would then have to be adopted. The medical officer must at all times be watchful in ensuring that individual men or units do not suffer from a lack of intelligent use of the facilities provided.

DIET.

The kind of food which is eaten has a considerable influence on heat production, and overfeeding or an ill-chosen diet may result in bacterial decomposition in the bowel with disturbance of the heat-regulating mechanism, and lowering of the resistance to heat. In adverse climatic conditions, the heat-regulating mechanism, already perhaps strained to its utmost, may be gravely embarrassed by having to deal with the added/

added requirements of a large meal rich in proteins which would cause blood to be diverted from the skin as a heat-eliminating organ[65] and this has already been referred to as a possible cause of circulatory failure. Simpson[244] believed that excess of meat issued to the troops in the South African war had some influence in causing Effects of Heat, and in a discussion on the occurrence of Heatstroke in Mesopotamia, Cope[293] suggested that their greater consumption of meat might account for the higher incidence of the condition amongst British soldiers than amongst Arabs and Indians.

In a series of 158 cases of Heatstroke reported by Gauss and Meyer[294] most were meat eaters. Hill[177] quotes Rubner's work on the specific dynamic energy of food stuffs, which showed that in an environment where heat-loss is impeded, or work so great that the body is in danger of overheating, carbohydrate is a better source of energy than protein, as it is less heating and increases the basal heat-production less. Cane sugar for example, is almost all completely used for work and has almost none of the heat-producing action of meat in the case of which the increased heat production due to the specific dynamic action of the meat protein is added to the heat produced by work which has to be got rid of by the body. In fat metabolism too, more energy is converted into heat than in carbohydrate metabolism. A diet which would favour the devel of obesity must be avoided as this condition greatly interferes with heat loss, increases the body's bulk relative to its surface area, and is generally recognised as predisposing to Heatstroke. Eijkman[(263)] however, found that food requirements with similar work are about the same in a hot as in a temperate climate and it is obvious that for muscular workers any reduction in protein intake must not be carried too far (89) (177) (251).

The need for an adequate supply of water has been indicated when describing the amounts of fluid lost by sweating, and some of the facts already mentioned may be recapitulated. Martin[(21)] showed that in hot climates ten large calories per minute may have to be removed from the body which would require the evaporation of a litre of water per hour. Other writers have shown that sweating may be so profuse as to necessitate the daily replacement of from five to six pints under ordinary circumstances up to twenty five or thirty pints with hard work and adverse climatic conditions (32) (56) to (58) (62) (150). The general symptoms caused by exposure to hot environments were much less severe when water was drunk than when it was withheld (32) (62) (103). Lee and Mulder[(63)] recently confirmed the beneficial effect of the intake of water upon human reactions to reduced cooling powers. They found that maintenance of body hydration markedly/

markedly increases the stability of the thermal equilibrium, and reduces the degree of shift of the equilibrium point, possibly largely on account of the increased rate of sweating. That circulatory functions are rendered more efficient by hydration is shown by the increased stability of the cardiac rate, less deviation from the normal and fewer symptoms due to poor circulation. Although the output of urine is increased by water intake this does not lead to undue loss of chlorides, since urinary chloride excretion is reduced during exposure to heat whatever the urine volume may be. There can therefore be no doubt that in hot climates the supply of drinking water should not be restricted, and an insufficient supply is placed by Willcox[(159)] and Mackenzie and Le Count[(246)] amongst the most important predisposing causes of Heatstroke. A further advantage of copious water drinking is that it may help to prevent a tendency to constipation which as Hurst[(295) (296)] says is apt to be caused by excessive loss of water in sweating, or by insufficient consumption of water. Constipation is often cited as one of the predisposing causes of Heatstroke (159) (223) (297), and in some series of cases it has been almost invariably present (225) (298) but Hamilton[(225)] doubts whether it should be regarded as a predisposing cause or a symptom. Constipation is common amongst soldiers[(295)] and I have/

have never been able to satisfy myself that it made them more liable to suffer from the ill effects of heat. A young soldier aged 20, who suffered from obstinate constipation, and who, if periodical treatment for its relief was withheld had no motion of the bowels for more than ten days at a time, felt and looked extremely healthy. He had a ruddy complexion, and proved that his thermal equilibrium was sound by regularly going for long cross country runs in very hot weather even when markedly constipated. That intestinal autointoxication is associated with constipation has been shown by Alvarez[(299) to (301)] to be more than doubtful, and if constipation has a bearing on the causation of Heatstroke, it may be as Hill[(177)] suggests that it is a sign of loss of tone and heralds the failure of the heat-regulating mechanism.

Most authorities are agreed that alcohol, especially if taken in the heat of the day or to excess, predisposes to Heatstroke (150) (154) (159) (168) (177) (297) (302) (303). Alcohol had been taken by all but two of the 158 cases reported by Gauss and Meyer,[(294)] and Talbott[(82)] found that it increased the tendency to Heat cramps. Pembrey[(224)] stated that alcohol causes diminished efficiency in temperature regulation, the normal reaction to heat and cold being blunted or even paralysed by large doses of alcohol. Whilst some of the lowest temperatures on record have been found/

found in drunkards exposed to cold, the disordered temperature regulation produced by frequent drinking may show itself in a high temperature on exposure to hot moist and stagnant atmospheres.

CLOTHING.

The wearing of unsuitable clothing impeding the free dissipation of heat throws a heavy strain on the heat regulating mechanism, and is a potent predisposing cause of Heatstroke (2) (150) (168) (177) (224) (225) (302) (304). Its importance is emphasised by Mouriquand[178] in his expression "Coup de chaleur vestimentaire". As Martin[21] says the chief obstacle to heat loss in the European is his clothes, and unless these are very thin and wet, much of the transference of heat takes place too far from the skin capillaries in which is the blood to be cooled, and a proportion of the heat absorbed will be derived from the air and surrounding objects instead of from the body. Although one effect of clothing is to increase the surface area, and so the effective radiating surface[305], the clothing worn by men in temperate climates with its inadequate ventilation at neck, wrists, and ankles, (made still more inadequate when breeches fastened at the knee are worn) is an effective barrier to the free circulation of air so necessary for the loss of heat. Indeed its effect is/

is to enclose most of the body in an artificial tropical climate with a temperature of from 87° to 91.5°F (263) (306) (307). An American reviewer(308) recently drew attention under the apt title of "The Fair and Warmer Sexes" to Dr Ernst Friedberger's remark that "the average modern man spends most of his life winter and summer in the debilitating climate of the Tropics. Only his face and hands are allowed to stick out into healthier surroundings.

The average woman on the other hand lives in a climate like the cool dry air of the Alps". Unsuitable clothing has been especially stressed as a common cause of Heatstroke in soldiers, (2) (177) (224) (304) but, whilst it must be admitted that the uniform now worn in temperate climates does, if we exclude the kilt, possess all the disadvantages above referred to, it will be seen when discussing the best type of clothing for tropical wear that the soldier abroad is provided with suitable clothes. Unintelligent use of this clothing must of course be prevented if we are to avoid cases of "Coup de chaleur vestimentaire". Such a case was that of a 16 year old band boy who went down with Heatstroke in a troop train crossing the Sind desert in April. He was wearing under tropical uniform of khaki drill, a thick woollen cardigan, flannel shirt and woollen vest. Although he had been given his drill uniform in the vicinity of Port Said on/

on the way to India, it had been left to his own initiative to make the appropriate changes in his underwear. His outward appearance was that of a soldier correctly dressed for India, but his discomfort during the journey, which included a very hot passage through the Red Sea, culminating in the breakdown of his thermal equilibrium in the Sind desert, can easily be imagined.

. . . .

PHYSICAL EXERTION.

Severe muscular exertion has already been mentioned as a cause of circulatory breakdown (p.53), and there is no doubt that exertion, especially during the heat of the day and if heavily loaded predisposes to the ill effects of heat. (150) (159) (168) (224) (302) (309). In Gauss and Meyer's[294] series 64.9% were labourers, and, despite the modern need for "brains" rather than mere "brawn", and the growth of mechanisation, the soldier is largely a muscular worker, and in time of war may have to undergo severe exertion under trying climatic conditions. In order that he may carry out his work with as little expenditure of wasteful energy as possibly, careful investigations have been made into various aspects of it, such as the type of pick to be used for digging and the best method of using it,[310] and the maximum load which he should be expected to carry and how he should/

should carry it[311], whilst the soldier himself is trained by a system of graded physical exercises until his mechanical efficiency is as high as possible.[312] Little supervision should therefore be needed to ensure that this factor makes no avoidable contribution to our cases of heat effects, in peace time at least.

PYREXIAL CONDITIONS.

Any pyrexial disease may, in hot weather, make the patient prone to develop Heatstroke, and the occurrence of the condition has been recorded in cases of Malaria, Sandfly Fever, Typhus, Enteric, and Smallpox, and the list could doubtless be added to. (150) (159) (160) (298). It needs no stretch of the imagination to believe that the heat-regulating mechanism, already unbalanced by the infection, is less able to withstand adverse climatic conditions, and there seems to be little justification for attempts which have been made to throw doubt on the existence of Heatstroke as a clinical entity, and to ascribe its clinical picture entirely to Malaria or to Sandfly Fever. Milner[313] denied the existence of Heatstroke and believed that the condition could always be attributed to the action of the parasite of Malignant tertian Malaria on the heat regulating centre. The frequent association of Heatstroke with the presence of Plasmodium falciparum in the blood cannot be doubted/

doubted,[223] [314] and probably many hyperpyrexial and comatose cases of Cerebral Malaria have been wrongly diagnosed as Heatstroke[71], but cases in which the two conditions might have been confused have occurred in patients who could not have been infected with Plasmodium falciparum[315], and Heatstroke of course is seen in patients who have never been in malarial countries. Sinderson[316] made a similar attempt to attribute all cases of Heatstroke to Sandfly Fever, but the refutation of this theory is on even sounder ground, since Sandfly Fever has a short incubation period, and does not, like Malaria, persist as a latent infection. Cases occurring where there are no sandflies, or in those on board ship en route for a first visit to the East, can hardly be due to Sandfly Fever. It is easy to believe that, as Morton[160] suspects, many cases of so called Heatstroke may have resulted from the effects of high atmospheric temperature and exposure to sun in undiagnosed cases of Malaria, Sandfly Fever, Enteric and other toxaemias, but to deny the existence of Heatstroke on this account is unreasonable.

Equally unacceptable is the theory suggested by Sambon[317], with whom no less an authority than Manson[318] agreed, that the condition is a specific infection and not due to heat. This view was upheld in a discussion in 1899 in the Tropical Diseases section/

section of the British Medical Association by Manson, Sambon, and Rho, but was opposed by MacLeod, the Professor of Military Medicine, and by other army medical officers[319]. The rapidity with which in many cases, all the symptoms of Heatstroke are relieved, and the condition completely cured by reduction of the temperature by physical means alone, suggests that bacterial invasion can hardly be the cause of the disease. The suspected organism was never isolated, and in the light of modern knowledge this specific infection can be regarded as mythical, and so at least to have been appropriately named 'Siriasis', a name for Sunstroke which emphasised its connection with Sirius the Dog Star whose appearance was of baleful import to the Greeks and Romans on account of its association with the late summer hot weather.

Apart from pyrexial conditions debility from any other cause will diminish the efficiency of the nervous regulation of temperature and so predispose to Heatstroke.[224] [297] A case was recently described by Fog[320] in which Paratyphoid Fever was followed after some years by a condition of anhidrosis similar to the congenital form already referred to, which like any other condition interfering with free sweating causes a tendency to Heatstroke.

AGE/

to Heatstroke, which may be accounted for by their diet of lower calorific value,more suitable clothing, abstinence from alcohol, less exertion and lower metabolic activity. They are usually of slender build, the surface of their bodies being relatively larger in proportion to their bulk than those of heavier build which is an advantage for heat loss. (150) (263). Although unacclimatised newcomers are very susceptible to the condition, long residence in the Tropics eventually neutralises the influence of acclimatisation by causing gradual deterioration of health, to which is added the factor of advancing age.

GENERAL EFFECTS OF HOT CLIMATES.

Heatstroke and allied disorders, though not of course confined to tropical countries are much more common there, and the description of the various factors which may sow the seeds of these conditions must be followed by a consideration of the soil in which the seeds may fall - the white man in hot countries.

A certain amount of deterioration in physical and mental health in Europeans living in the Tropics is so common that Drinker[(251)] and Black[(322)] regard two or three years as probably the longest period of residence consistent with safety. Growth and physique are apt to be impaired and "weedy" long-legged types are common, which is to some extent a wise provision of Nature since the tall thin type is best suited to tropical life having the maximum surface for heat loss in relation to body weight. Minor differences in the growth of skin, hair, and nails, are recorded (169)(248).

Growth and physique are especially affected in women and children[(169) (251)] and Rattray (323) (324) found that naval cadets between 14 and 17½ years grew too quickly, and lost weight considerably, and that their strength and health were greatly impaired by heat. The lack of change is an important feature of tropical climates and in long spells of hot weather the/

the body has no opportunity of reacting to cold and so loses the power of doing so, and with long exposure to such conditions the body becomes enfeebled and its resistance to disease is lowered. Mental energy too is sapped and the individual becomes fatalistic, and lacking in industry and inclination to work, with forgetfulness and inability to concentrate. Irritability is often marked and the individual is apt to give way to bursts of violent anger in circumstances which at home would give rise to mere vexation, a condition sometimes called Furor tropicus [(150) (169)]. Those who have worked in hot countries will agree with Huntington[(325)] that much of this irascibility arises from attempts to hustle the slow tropical people, and the fact that a show of violence is often the only way to get things done is made an excuse for a loss of self control. Further the consequences of temper are often less dangerous as the inert dwellers in the Tropics will put up with more abuse than would the more alert Northerners. The impairment of mental efficiency and weakness of will power, combined with the added "pinpricks" of fatigue from the heat, the glare of the sun, insect bites, "prickly heat," loss of sleep and visual disorders predispose to Psychasthenia or Neurasthenia which is common in the Tropics. (169) (262) (263) (326) (327) (328). The condition/

condition is sometimes called Tropical Neurasthenia but although Neurasthenia is more common in the Tropics than in cool climates Megaw[150] and Culpin[329] point out that there is no justification for regarding it as a tropical disease since there is no evidence that any form of Neurasthenia is peculiar to the Tropics. The causes of Neurasthenia in the Tropics are the same as they are in temperate climates, with the addition of depressing environmental factors some of which have been mentioned, and the toxins of such diseases as Malaria and Typhoid, which Henderson and Gillespie[330] include amongst the causes of Korsakow's Psychosis. Latham[331] believes that with increasing altitude psychological effects are more common although the incidence of tropical diseases is less.

White men who spur themselves to work as hard in the Tropics as at home risk a breakdown in health, and undue fatigue may lead to fatigue indigestion and a resulting physical depression[164] [325]. The causation of fatigue is perhaps imperfectly understood, and as Eijkman[263] observes, the toxic action of products of metabolism cannot account for early morning fatigue. When the cooling power of the air is inadequate the amount of blood in the skin leads to a decreased supply to the muscles, but the process by which this inadequacy finally manifests itself as a feeling of exhaustion is still/

still unknown.[11] The glare of the tropical sun has been described as a common cause of Hemeralopia in soldiers, and the part played in this by vitamin deficiency in the diet must not be forgotten[332]-[334]. The glare also causes headache and other minor visual disturbances whilst trifling errors in refraction cause effects out of proportion to their severity[164] [326]. Fatigue of accommodation is common in the Tropics and often follows illness, toxic conditions and Neurasthenia, whilst superficial irritation, Conjunctivitis, and lachrymation with frontal headache are not uncommon.[326] [335].

A slight increase in body temperature is often noted[4] [251] but Castellani and Chalmers[169] who quote a number of authorities who record this fact, did not themselves find any alteration in the temperature whilst Wick[336] thought that there was no increase in body temperature if the rise in environmental temperature was gradual. During hot weather in India one often found the normal temperature to be above that usually regarded as normal whilst in cold weather it was often considerably below that figure especially in the morning. In this connection it may be noted that Murray Lyon[337] and Paton[338] consider that in temperate climates 97° or 97.4°F is the usual mean temperature of afebrile patients and that a rise to 98.4°F may indicate some pathological disturbance.

Pallor/

Pallor is so common that many have regarded it as being due to a so-called "Tropical Anaemia", and Megaw(150) quotes Maurel's views that in Europeans going to the Tropics there is at first stimulation of the blood forming organs which is soon followed by exhaustion, so that the red blood corpuscles fall to 3½ to 4 million with anisocytosis and polychromatophilia and a proportionate reduction in haemoglobin. The chief manifestations of this anaemia are said to be mental and physical weakness, pallor and palpitation, oedema of the feet and emaciation. Others have not confirmed these findings and have found the red blood corpuscles and haemoglobin normal or increased (240) (248) (251) (263) (336).

Eijkman(263), who found blood regeneration after haemorrhage to be normal, says that in temperate climates the covered parts of the skin may be in an artificial tropical climate and are pale and the red parts of the skin when in a warm climate similarly become pale. The colour of the face is largely due to wind and weather and the pigmentation of sunburn, and it seems probable that despite tropical pallor haemopoiesis is not impaired and that there is no purely physiological anaemia of hot climates. Certainly one must agree with Megaw(150) that a diagnosis of Tropical Anaemia should never be made until all other causes have been excluded, remembering especially/

especially Malaria, Ancylostomiasis, Dysentery, Syphilis, and Cancer.

The number of white blood corpuscles is normal or decreased and there may be some eosinophilia (248) (251)(339). The Arneth polynuclear count shows a shift to the left, and the Arneth Index which is normally 2.62 to 2.75, or 37 to 43 according to the different ways of estimating it, (340) to (343) may be 1.94 to 1.98 or 71 to 83.86 (344) to (347). In a hundred healthy soldiers in the Plains and after proceeding to the Hills in India I found that the average count showed a shift to the left but of a less marked degree than those quoted above, the average index being 2.59 or 45. There was no significant difference between the counts taken in the Plains and those taken at an altitude of some 7000 feet above sea level, such as that recorded by Kennedy and Flint[(348)(349)] in the Alps and possibly due to the influence of ultra violet radiation. Shanklin[(350)] similarly found that amongst the Alouites in Syria who have the lowest recorded index of any racial group, there was little difference between plains and mountain dwellers. Malaria and Yellow Fever cause a marked shift to the left[(351)] and I found a considerable shift to the left also in Dengue and Sandfly Fever. Whilst therefore latent infections may be of importance in producing the more marked variations of the count it is generally held that/

sedentary habits, whilst in labourers Talbott[85] found a slight increase in blood lactic acid.

Opinions on the pulse rate and blood pressure vary but the changes recorded are for the most part only slight ones (85)(169)(240)(248)(249)(251)(263). Roddis and Cooper[356] however found the blood pressure of natives of tropical countries to be low, and that of Europeans to be 10 or 15 millimetres below the normal for temperate zones, and Treadgold[357] found that although moderately warm climates have little effect on the blood pressure, very trying tropical climates cause a definite fall in blood pressure values as a whole. I found no significant changes in the blood pressure of healthy soldiers in India, in the Plains or in the Hills, but the readings tended to be low rather than high.

The respiration rate is usually somewhat slow, with an increase in capacity.(169)(251)(336)(358).

The volume of the urine may be slightly decreased, and its specific gravity raised, but after adaptation there is little difference in volume from that in temperate climates, and the specific gravity is normal (85)(169)(251). The excretion of nitrogen is reduced probably on account of the low protein intake(85)(359). Digestion is apt to be somewhat impaired, and there is lessened appetite especially for meat, and a tendency to constipation especially if insufficient water is/

is drunk. Digestive upsets are all too apt to be laid at the door of the liver, at least by the sufferer, and often by a too complacent medical adviser, and many who have lived for long in the Tropics consider it their privilege to attribute most of their ailments to this organ, and are encouraged in so doing by the use of the term "Tropical Liver", especially popular amongst French writers. Megaw[150] believes that the Liver is "more sinned against than sinning," and that there is little evidence of disturbance of liver function in those who lead rational lives. Those who take too much meat or alcohol, and too little exercise, must expect derangements of the alimentary system including the liver. Gastro-intestinal catarrh with absorption of toxic substances may damage the liver, and in such cases search must always be made for evidence of Amoebiasis, Syphilis, and other causes of hepatic disorders.

Amoebiasis deserves special mention, for it is a frequent and often undiagnosed cause of digestive disorder and of general ill-health. It is not by any means exclusively a tropical disease, and is more common in temperate climates than is generally realised (360) to (363). It is much more prevalent in hot countries (364) to (366), and in some parts of India has been found in some 10 to 15% of natives and a considerable proportion of Europeans (367)(368). When/

When it was realised what a high percentage of people in England were infected with Entamoeba histolytica doubts were expressed of its being the true pathogenic form, but Dobell[360] held that it certainly was. These doubts based as they were upon the absence of symptoms emphasise the important fact that amoebic infection may be entirely symptomless. It is most important to realise that the Entamoeba histolytica may cause not only typical acute amoebic dysentery which may from ineffective treatment give rise to chronic amoebic diarrhoea, but may cause a mild infection from the outset. In such cases there may be a history of a passing attack of acute diarrhoea which is soon forgotten, or it may be impossible to extract any history of dysenteric symptoms at all. Often there is only a tale of general ill-health with no bowel symptoms except perhaps for a tendency to constipation or an excessive reaction to purgatives. In other cases gastric symptoms may predominate, and there may be reflex hyperchlorhydria, flatulence and indefinite pains in various parts of the abdomen. Excessive sweating, low grade temperatures, extreme fatigue, and unexplained loss of weight may suggest Tuberculosis, and the infection may also cause a clinical picture very hard to distinguish from Chronic Appendicitis (369) to (375). In cases such as these a biological balance has been reached between the host and the amoeba/

amoeba who has established himself in a few latent amoebic ulcers from which, when the vitality of the host is lowered or perhaps when a superimposed bacillary infection occurs, he may spread to fresh areas. This latent Amoebiasis often remains unsuspected until perhaps a Liver Abscess develops, and Megaw[150] could find no history of previous dysentery in 20% of admissions for Liver Abscess, although on post-mortem examination a few latent amoebic ulcers were almost invariably found.

The debility due to such latent Amoebiasis may be a frequent unsuspected cause of predisposition to Effects of heat.

Another possible cause of digestive upset and ill-health is suggested by Kligler's[376] observations on the intestinal flora in the Tropics. Various conditions cause alterations in the intestinal flora and definite standards are hard to fix[377][378] but Kligler's opinion is that the reduced cooling powers of hot humid climates result in a retardation in gastric secretion and so a breaking down of the natural barrier to infection. Excessively dry hot climates may produce the same effect by causing a loss of water, the anhydraemia thus produced reducing stomach tonus and gastric activity and increasing susceptibility to enteric infections. The same result may be brought about indirectly by the effect of high environmental temperatures on nutrition, with inadequate/

inadequate intake of Vitamin B, loss of appetite, and a state of B avitaminosis, and these effects of a hot environment are increased by direct solar radiation. The antiseptic qualities of the gastric juice are probably of great importance[379][380] and Blacklock[381] considers that the essential factor in acute Enteritis in children is a breakdown in the normal physiological function including the level of gastric acidity which keeps the upper part of the highly absorptive small bowel relatively sterile. Sir Arthur Hurst[382] thinks that the fractional test meal should, as advised by Camps[383], be an essential part of the examination of soldiers and civilians before serving in the East, and that achlorhydria which he believes would be discovered in about 4% should be regarded as an absolute bar to such service.

Skin affections are common in the Tropics, as the skin flushed with blood and wet with sweat in the damper parts of the body, is liable to fungous affections such as Prickly Heat and various forms of Tinea, whilst in hot dry climates the skin is dry, and liable to become inflamed and cracked. The prevalence of skin affections, the importance of which as a cause of external Otitis was recently stressed by Brown[384], may contribute to the high incidence of Otitis for which bathing parades are often blamed as a contributory cause[385][386]. The irritation and sleeplessness/

sleeplessness due to these skin conditions contribute to the general ill-health and are added to by insect bites. Benson[387] believes that allergic manifestations may result from sensitization to the products of mosquitoes which have their avenue of entry by the insect's sting.

ACCLIMATISATION.

Although a somewhat depressing picture of Tropical life has been painted by this enumeration of the ills the flesh is heir to in hot climates, most of us manage to remain not only healthy but very happy there. Whilst it is certainly inadvisable that European children should remain too long in the Tropics, one can hardly accept the limitation of an adult's stay to two or three years advised by Drinker[251] and Black[322]. Soldiers and many others must and do remain for much longer periods, and most of them do not suffer unduly in health. They must of course adjust themselves to a somewhat different method of living. The native of tropical countries is protected against the injurious rays of the sun by pigment, and the white man can and should acquire this protection. The native is less liable to excessive sweating and here again we have seen that acclimatisation is possible. The acclimatised white man sweats less profusely, and loses less salt in the sweat, thus avoiding wasteful dehydration/

dehydration and salt-depletion (3)(69)(84)(85). Training can develop the power of heat regulation, and effect the production of work with the least amount of body heat.

In all these respects acclimatisation of the white man in the Tropics is possible, and whilst these physiological adaptations, which take some time, are occurring, he should be learning to adapt himself to the climate in such matters as habits and clothing on the lines which will be discussed when dealing with the Prevention of Heatstroke.

CLINICAL CONDITIONS DUE TO FAILURE OF ADAPTATION TO HOT ENVIRONMENTS.

The clinical conditions due to heat will be dealt with in terms of the four types of failure in the adaptation of the body to heat which have been described, although it will be obvious that these cannot be regarded as separate and distinct entities and that there are no hard and fast lines between them. Dehydration and Salt Loss are often combined, and are conducive to the development of Hyperpyrexia, which itself is often associated with Circulatory insufficiency. Circulatory insufficiency often occurs in those who are suffering from minor degrees of Salt Loss. Circulatory insufficiency or Heat Exhaustion and Hyperpyrexia or Heatstroke proper may merge into one another, so that a severe case of Heat Exhaustion can often be regarded as a case of Heat Hyperpyrexia in the prodromal stage, whilst an attack of Heat Exhaustion with low temperature may occur during the course of a case of Heat Hyperpyrexia. A consideration of which of these mechanisms is the predominant one in any case may afford useful indications as to the treatment which should be undertaken. Dehydration and Salt Loss, which in their purest forms are seldom encountered in peace-time military practice will be dealt/

dealt with first and finally the clinical features, treatment and prevention of Heat Exhaustion and Heat-stroke will be described.

DEHYDRATION.

In cases associated with excessive loss of fluid by sweating, especially in hot dry climates there is always a risk of the development of dehydration, and Mouriquand(178) finds that, in children especially, very severe and rapid dehydration may occur. Stott(175) compares the clinical result of dehydration with that of an attack of cholera. There is loss of weight and the skin becomes grey, wrinkled, dry, and inelastic and sometimes cyanosed. The mucous membranes are dry, saliva reduced, and the lips and tongue shrivelled. The tongue is usually coated and foul. There is dysphagia, anorexia, and severe thirst. Muscular weakness may be extreme and nervous and mental disturbances include sensory disturbances, sometimes anaesthesia, irritability, drowsiness and stupor. The temperature is usually normal though in the grave dehydrated form of Mouriquand's(178) "Syndrome du Vent du Midi" there is usually hyperthermia. A terminal rise of temperature to 104°F or more may occur, and even when the temperature is high the extremities are cold. The pulse is feeble and of low tension, but the blood pressure is well maintained possibly on account of the increased/

increased viscosity of the blood. The diminution in blood volume is associated with functional electrocardiographic abnormalities such as absent T waves, abnormal Q.R.S. complexes and increased P.R. intervals, which disappear when the blood volume returns to normal[73]. The impaired pulmonary circulation may cause intense dyspnoea, and minor bouts of Cheyne-Stokes breathing sometimes occur, whilst acidaemia may result in the deep breathing of air hunger. The secretion of urine is much reduced, and a few ounces only may be voided in the 24 hours, and that of a deep colour, intensely concentrated and sometimes so acid that its passage is associated with a slight sting[388]. Terminal convulsions may occur, possibly in association with uraemia which is often present[73]. The condition of those suffering from the most advanced stages of dehydration was described in 1877 by King[388]. An American patrol was lost in the Texas desert for three and a half days with no water, and he describes their aged and careworn faces, their mouths and throats so parched that they could not even dissolve brown sugar in their mouths and could not swallow it. They drank the viscid blood of dead horses and sucked their hearts and viscera, and had to hold the clotted blood for some time in their mouths before they could swallow it. The intense thirst which as Steevens[389] said causes the feeling that "your muscles shrivel to dry/

dry sponge, your bones to dry pith" cannot be relieved until the remotest tissues are supplied. The Texas patrol suffered also from vertigo, dim vision, deafness, delirium, and mutual suspicion and lack of confidence, and could only move with a feeble and tottering gait. On account of the impairment of the pulmonary circulation from anhydraemia although their Lungs "were filled with the purest air, yet they appreciated an almost overwhelming sense of suffocation."

The laboratory findings in dehydration have already been described (pp. 55-60): they consist essentially of evidence of blood concentration, a rise of blood urea and impairment of renal function, and varying disturbances of the acid base balance, acidaemia or alkalaemia. According to Maizels and Smith[77] acidosis with a low ammonia coefficient, or alkalosis with an acid urine, are of bad prognostic significance. The degree of acidosis alone is no guide to the severity of a case, for those with marked acidosis may recover,and those with a normal alkali reserve may die.

TREATMENT.

The obvious necessity is to restore the body fluids, and the patient should be encouraged to drink/

drink freely, and may be given water, or sweetened tea, coffee, lemonade, and similar beverages. Absorption of fats and proteins in the intestinal tract is defective, and vomiting is apt to occur when food is given to subjects in advanced states of dehydration, and fluids also, if too greedily drunk, may be vomited. Mackay[75] found that convulsions resulted from giving water or saline to severely dehydrated animals. Fluid must be given also by other routes, including the useful one for dehydrated children - by intraperitoneal injection, until the dehydration is relieved, and subcutaneous and intravenous injections of fluid are especially important of course in the case of severely dehydrated patients unable to retain fluid given by the mouth. Maizels and Smith[77] and Hartmann[390] consider that normal saline is a bad fluid to give in dehydration since it may increase any tendency which exists to acidosis, especially if the blood chloride be high as it sometimes is (p.57,58.). This is 57 58
because the sum of chloride and bicarbonate in the blood is fairly constant, and as the one increases the other decreases, so that if a strong solution of saline is injected the kidneys excrete not only chloride but bicarbonate and thus the alkali reserve may be lowered. Furthermore as the kidneys endeavour to excrete the excess of chloride and as the concentration of chloride in the urine does not usually exceed 0.45% much water must be lost with the chloride, and so the/

the dehydration may be actually increased. Hoag and Marples[391] did not find any increase in blood chlorides or any acidosis after saline injections, but they gave also copious intravenous injections of glucose. If saline therefore is given it should be 0.45% hypotonic saline, and may be combined with 2½ to 5% of glucose. If acidosis is severe, glucose may be insufficient and then Hartmann[390] advises that sodium bicarbonate should also be given as in these circumstances the risk of alkalosis is slight. Marriott[73] believed that sodium bicarbonate may be harmful, as it has the effect of further concentrating the blood, and because of the alkalosis which often occurs with the reëstablishment of a normal water balance, and he found that it might lead to the occurrence of tetany. The solution of saline and lactate advised by Hartmann[390] [392] is useful, as it is neutral and does not disturb the reaction of the blood, whilst if acidosis is present the lactate is converted into bicarbonate. If however acidosis is not present the conversion of the lactate into bicarbonate is so slow as to cause little or no risk of alkalosis, so that it can be used for the relief of dehydration regardless of whether acidosis or alkalosis is present. Mouriquand[178] adds to the solutions for intravenous injection one or two units of insulin which helps to keep the fluids in the tissues. When dehydration has existed for some time and water is not restoring the blood volume, blood transfusion is helpful.[73]

SALT LOSS: HEAT CRAMPS.

Minor degrees of salt loss are as we have seen probably quite common in the Tropics (p.66-67; 80-81) and may be suspected if the urine does not contain the normal amounts of chloride. The symptoms which may be associated with such salt depletion include some loss of weight, undue fatigue and lassitude, headache, sleeplessness, lack of concentration, loss of appetite, soreness, stiffness, and twitchings of muscles and excessive sweating. (20)(58)(98)(99). Minor attacks of muscular cramp are not uncommon in cases of Heat Exhaustion and Heatstroke, but they are seldom so severe as the typical Heat cramps occurring amongst workers in hot environments such as mines and stoke holds. The relation of this condition to Heat Exhaustion and Heatstroke is not clearly defined. Both follow exposure to hot temperatures, and the resultant loss of large amounts of sweat, and the possibility that salt loss by depressing sweating may predispose to Heatstroke suggests the possible link between them of a common causal agent. Talbott[86] suggests that a point against a common cause is Willcox's[159] observation that convulsions in Heatstroke are made worse by intravenous saline, which immediately relieves Heat cramps. The performance of hard work is a more or less essential factor in the causation of Heat cramps, whereas of course Heatstroke can occur without this factor. Although these conditions have

a/

a similar point of origin further comparison makes it clear that Heat cramps should be regarded as a clinical entity and not as a type or as a symptom of Heatstroke.

Individual susceptibility to cramps varies; Edsall[393] described a case in which a tendency to myospasm had existed from childhood.

Acclimatisation is important especially with regard to the amount of sweat lost and its salt content (p.64). Predisposing causes are ill health, alcoholism, inadequate assimilation of food, poor hygiene, and recent attacks of untreated cramp.

Vague prodromal symptoms may occur, such as mild vertigo, headache, and feeble twitchings of the muscles. Pain or tingling in the muscles or in the fingers and toes may precede or accompany the cramps.[394] Any muscles or groups of muscles may be affected, especially those most used at work, and groups affected in a first attack are likely to be affected in subsequent ones. The calves, flexors of fingers, and forearms, and abdominal muscles are commonly involved but many groups may be affected, and sometimes the cramps attack one muscle after another, sometimes sparing the small muscles they attack the larger ones. The vomiting and diarrhoea which may occur have been attributed to an effect on the smooth muscle of the gut.[86] Electrocardiograms are normal and Talbott[89] thinks that myocardial spasms are unlikely. The time /

time of onset varies, and the cramps may be started by a cool breeze, cold water, or jarring of the bed, and palpation may cause violent contractions. The pain may be very severe indeed, and is sometimes excruciating,[395] and voluntary use of the muscles during contractions is usually impossible. The muscles may be stony hard, the contractions are not relieved by firm pressure, and fibrillary twitchings may be seen[393][396]. As improvement takes place, the free period between cramps lengthens.

Clinical examination is more or less negative. The temperature is normal or shows a slight rise, and the pulse rate also normal, or if accelerated is seldom over 100. The blood pressure may be below normal but is sometimes raised. The deep reflexes may be temporarily exaggerated or depressed, and the pupils, which react to light and accommodation, are not persistently contracted or dilated, but sometimes dilate with each cramp. The oliguria recorded by Lee,[20] which one might expect on account of the abundant sweating, is not confirmed by Talbott,[89] who states that large amounts of urine are voided with a pH of about 5·0, though the excretion of titratable acid is not much increased. Although a small amount of sodium chloride may be present in the admission specimen, it is later absent, and in severe cases may continue to be so for some days. Phosphaturia and creatinuria are often observed, and the /

the excretion of total nitrogen is usually increased. Albumin and casts are usual in the admission specimen.

The essential feature of the condition shown by blood analysis is hypochloraemia, the chlorides being reduced on an average by 4 to 10%,[97] and there is evidence also of anhydraemia, an increase in the plasma proteins and haemoglobin. There is considerable increase in the serum calcium, and non protein nitrogen and blood sugar may also be increased.[89] We have seen that salt loss is usually associated with alkalaemia (p.79), and this is the usual but not invariable tendency of the acid base balance in Heat cramps[20][89] Cases in which acidosis have been recorded were probably associated with a considerable degree of dehydration. There is a leucocytosis with a relative increase of polynuclear leucocytes.

Few autopsies are on record, and the only features noted have been contraction of the left ventricle and congestion of the Lungs, and of the gastric mucosa.

DIAGNOSIS.

The occurrence of painful muscle cramps in men working in high temperatures and losing sweat profusely should suggest a diagnosis of Heat cramps. It may be difficult to distinguish the condition from the ordinary cramps which follow violent exercise, and nocturnal cramps, especially as Heat cramps may have a long latent period, and may not develop until some /

some hours after the cessation of work. It is possible that those more common forms of cramp may have a similar pathogenesis to Heat cramps. When the cramps are confined to the abdominal muscles the differentiation from simple colic may be difficult. The history and careful clinical examination will exclude such conditions as convulsions due to organic disease, to uraemia, hysteria or strychnine poisoning, and trichinosis, writer's cramp, or tetany from various causes. Milder cramps may be incidental to an underlying state of Heat Exhaustion or Heatstroke. The marked reduction or absence of urinary chlorides is a ready aid to diagnosis. As regards the blood changes, Talbott[89] points out that the only instance of a similar increase of calcium and of plasma protein is in Addison's disease. He finds the ability to produce the cramps, as for example by cold water, a useful aid to diagnosis and a test for cure.

TREATMENT.

The administration of saline solution is specific, and affords speedy relief, and a 0·1% solution may be given by mouth, subcutaneously or intravenously. In severe cases Talbott[89] recommends giving 600 to 1000 c.c. intravenously in the first six hours and repeating this amount if the patient is much dehydrated. After the first few hours the patient can begin taking salt by mouth. The addition of sodium /

sodium bicarbonate to the saline, which has been recommended, is not necessary and may be harmful as severe alkalosis may be present despite an acid urine.[73][89] A milk diet should be given and the patient kept at rest which may in the milder cases be enough to bring about recovery. Hot packs may give some temporary relief. Fantus[397] recommends the giving of morphia, or chloral and bromide, or even chloroform, to produce muscular relaxation if the cramps are very severe. Talbott[89] finds that morphia does not stop the cramps nor relieve the pain, and says that such remedies as calcium-chloride, amyl nitrite, and chloroform are of historic interest only.

PREVENTION.

Measures should be taken to ensure good health amongst workers, and a satisfactory environment both on and off duty, plenty of sleep, good food, and milk are necessary. The diet may need adjustment but the restriction of meat advocated by some must not be carried too far.[89][251] The most important thing is to ensure that the daily supply of salt is greater than that lost in the sweat and that the urine is not allowed to become salt free. This may be achieved in a variety of ways, such as by giving salt meat or fish, salted beer, or saline solutions. (69)(86)(89)(90) (92) to (97). The saline solution should be about 0·1% as this concentration causes no perceptible saline /

saline taste and allays thirst whilst stronger solutions may cause thirst, may lead to water retention, or raise the serum chloride too high. The solution may be taken at meals, or salt tablets with directions as to strength may be given to the workers or left at drinking water taps. Dextrose has been found useful but although a useful source of energy it has no special value in preventing cramps. Oatmeal water found useful by Gardner[398] is of value only as one way of increasing the fluid intake. Cold baths should be avoided as they increase the tendency to cramps.[399] Individuals unduly susceptible to cramps whether from predisposition or from failure to adapt themselves to heat, may have to be excluded from the type of work which causes the condition. In all cases time should be allowed for acclimatisation, and workers should at first be given light tasks, their salt intake being liberal.[89][400] Further after a cool period of more than a few days duration similar precautions may well be taken as adaptation to heat is rapidly lost.

Heat cramps are not of great importance to the military medical officer who will probably seldom if ever see a case, and the foregoing account, much of which is derived from Talbott's[89] extensive survey of the condition, has been included chiefly in order that no aspect of the Effects of Heat should be neglected.

Of /

CIRCULATORY INSUFFICIENCY: HEAT EXHAUSTION.

Of Heat Exhaustion and Heatstroke on the other hand the military medical officer may, as indicated already, see many cases, and may encounter his first experience of them before his first tour of Foreign service during a hot summer at home, on board a troopship bound for the East, or in the troop train which carries him to his new station on his first day in a strange tropical country.

The typical case of Heat Exhaustion results from circulatory insufficiency, when the heart fails to respond efficiently to the strain of keeping the cooling system going in addition to the ordinary work required of the circulation. The condition cannot however be divided by any hard and fast line from Heatstroke proper, and many cases diagnosed and successfully treated as Heat Exhaustion could equally well be regarded as cases in the Prodromal stage of Heatstroke which have been saved from developing further. The common case of collapse from heat which is so often seen amongst crowds on a hot day, or in troops on the march may occur quite suddenly and without warning, but during spells of hot weather in the Tropics there is sometimes a fairly prolonged prodromal stage of some three to four days, during which there may be anorexia, weakness of the limbs, headache and constipation. There may be a history of inability to sweat, and a dry skin, lassitude and sleepiness for/

some days before the attack. Not uncommonly as Barclay[401] noted in 1859 an outbreak of cases of Prickly Heat precedes the occurrence of cases of Effects of Heat. The prodromal symptoms may be followed by sudden collapse. The symptoms of the condition are those of shock with giddiness, faintness, pallor, a cold moist skin, sweating, and prostration. The temperature is usually subnormal, sometimes after a transient initial rise, though Willcox [159] and Hamilton[225] describe a mild pyrexia of 102° to 103°F lasting for some days, a finding which probably entitles the case to be regarded as one of Heatstroke in the prodromal stage. In Fantus' [397] experience the temperature is rarely over 101°F. Nausea and vomiting occur, the latter being sometimes persistent and the vomitus eventually becoming bile stained.[160] The pulse is rapid and feeble, the blood pressure low, and some cardiac dilatation may be detected. Hamilton[225] found the most notable feature to be "cardiac irregularity coupled with haemic murmurs and in some cases slight cardiac dilatation". He felt uncertain whether this condition was entirely secondary, or a predisposing cause, and stressed the importance of routine examination of men before the hot weather. The common occurrence of myocardial degeneration in hot climates observed by Brown[170] has already been mentioned (p.93). The pupils are dilated and the reflexes present. Consciousness may be partially lost or /

or there may be temporary unconsciousness, and Heilman and Montgomery[97] frequently found relaxed sphincters and gastric regurgitation. Sensory changes have been described by Lee.[20] In one of Morton's[160] cases, although there was no hyperpyrexia, there was maniacal delirium but this was followed by no permanent mental changes. The respirations are usually shallow and sighing, but there may be hyperpnoea with alkalaemia. In more severe cases acidaemia may be present. Cramps may occur varying in intensity from slight abdominal colic to severe abdominal and other muscular cramps. There is less depletion of the blood chlorides than in Heat cramps, the average blood sugar is low, and Morton [160] found the blood urea slightly raised one month after recovery in one case, although in no instance was there any evidence of permanent renal damage.

Recovery is usual although in severe cases headache and postration may persist for some time. Sometimes death occurs from syncope, or the unconsciousness may deepen into coma, and death ensue. With early and appropriate treatment the symptoms usually quickly improve, though otherwise there is a risk of the development of Heat Hyperpyrexia.

On postmortem examination the heart may be found to be flaccid, softened, and full of blood especially on the right side. There may be some congestion of the Brain, Lungs, and other viscers, but generally there is little to note.

TREATMENT.

The patient should be removed to a cool place, made to lie down with his clothing loosened, and kept at rest. An aperient may be given. The limbs may be massaged, and a little water sprinkled on the face and chest, but the principle indication is stimulation. A little brandy or sal volatile may be given, or to patients unable to swallow, hypodermic injections of ether, or camphor, and ammonia may be applied to the nostrils. Fantus[397] recommends subcutaneous injection of 0·25 to 0·5 grammes of caffeine sodio benzoate every four hours, and if the pulse is very rapid, the intravenous injection of 0·5 mg. of Strophanthin which should not be repeated for 24 hours. He emphasises the necessity for supplying salt and water, and Lee[20] also advises the giving of saline and the promotion of the venous return by recumbency and bandaging or massage of the dependant parts.

Morton[160] gives the following mixture hourly for 24 hours - Glucose ʒi Sodium bicarbonate gr. XV. Water to ℥i , or Potassium citrate gr. XXV. If neither are retained he gives intravenously one to one and a half pints of 2% Sodium bicarbonate in saline sometimes with 5% of glucose which he finds gives dramatic results. Heilman and Montgomery[97] recommend beverages containing sugar and find sweetened lemonade or lemon juice the best, since it contains /

tains base linked with weak organic acids, and serves to strengthen resistance to depletion of the blood sugar and to acidosis.

When there is collapse and a subnormal temperature, the patient must be kept warm with hot water bottles, a warm bath may help, and hot drinks may be given, care being taken that a rise in temperature is not caused. Should the temperature rise above 102°F. it may be necessary to reduce it by cold ablutions as practised in cases of Heatstroke.

Any pre-existing disease must be taken into consideration, and receive appropriate treatment, and, after recovery the patient should be protected from exposure to adverse climatic conditions for some time and severe cases may well be given a period of convalescence in the Hills.

HEAT HYPERPYREXIA: HEATSTROKE.

Heat Hyperpyrexia may be regarded as the typical Heatstroke, and we shall include under the heading the very acute condition often referred to as Sunstroke. This is as Osler(302) says one of the oldest of recognised diseases, the case of the son of the Shunammite woman described in the Bible (II.Kings iv. 18) being probably the oldest on record. It is not surprising therefore that it has been given many names, and somewhat unnecessarily classified into various types.

The underlying process is essentially the same in all these types which result from a preponderance in certain cases of some of the pathological results and symptoms of the condition, and the addition to Hyperpyrexia of varying degrees of Salt Loss, Dehydration, or Circulatory insufficiency.

SYMPTOMS AND CLINICAL COURSE.

PRODROMAL STAGE:- The onset of Heatstroke may be very sudden but usually it is more gradual, and a distinct prodromal stage lasting from an hour or less to two or three days or longer may be distinguished. During this period the patient may be drowsy and disinclined for work, and may suffer from weakness, pains in the limbs, feelings of constriction in the chest, malaise, vertigo, and headache. Thirst is usually complained of and anorexia, nausea, and vomiting are common. /

common. Photophobia is often present, and chromatic aberrations of vision are also reported.(71)(169) Mental confusion, restlessness, and irritability or dullness are characteristic, and the patients are often said to be quite unlike their normal selves, and are sometimes disrespectful and insubordinate. These mental changes have actually led to soldiers being punished, and I have a vivid recollection of a rather painful scene in one of my wards in Allahabad. A young soldier whom I knew well to be normally of a cheerful pleasant disposition and a particularly courageous boxer, behaved like a spoiled child whilst I was examining him. The nursing sister impatient apparently of my efforts to win his confidence and to persuade him to let me examine him, and herself feeling no doubt a trifle "hot weathery", shook him by the shoulder remarking "That's no way to speak to an officer." This caused him to become quite hysterical, and no further progress could be made until the militarily-minded sister had left the ward, and the relationship of doctor and patient could be re-established, at the expense of that of officer and soldier!

A sense of horror or of impending calamity, sighing, and a hysterical tendency to weep have been noticed in some cases.(71)(402) The pulse rate may be rapid, the skin hot and dry, the eyes suffused and the /

the pupils contracted, and the face is usually flushed though it may be pale. Very commonly there is frequent passage of small amounts of urine and often some burning pain on micturition. This bladder irritability which is probably due to the highly concentrated and often acid urine is one of the most valuable danger signals. This prodromal period is usually but not invariably apyrexial, and it will be realised that in many of its features it resembles the condition of Heat Exhaustion, every case of which should be regarded as a potential case of Heatstroke. Whereas however many cases of Heat Exhaustion will recover spontaneously without any treatment other than rest, this is not always the case with those in the prodromal phase of Heatstroke, and this particularly applies to an important condition called by Willcox[(159)(252)] the "Gastric Type" of Heatstroke, which should I think probably be regarded as a prodromal stage of Heatstroke. The patient has a flushed face, is restless and irritable, and nausea and vomiting are marked. The mouth temperature and pulse are normal but the rectal temperature is often slightly raised. Willcox found usually a fatty enlargement of the Liver and noted that the Knee Jerk was absent as a rule which is a valuable diagnostic sign.

After anything from four to ten days of such a mild /

mild illness, the patient suddenly develops Hyperpyrexia which very often proves fatal. Of sixteen cases of this treacherous condition in India in 1932 nine died.(298) One of these Lance Corporal S. aged 29, was the only fatal case amongst twenty treated by me in Allahabad. He was detained in hospital suffering from mild gastritis for which he was treated on routine lines. Inclined to be morose and sullen his demeanour gave rise to the suspicion that his gastritis was partly due to alcohol for I knew him to be a moderately heavy drinker. He appeared to be progressing favourably, but whilst engaged in treating an acute case in the Heatstroke Ward I was surprised to learn that he was unconscious with a rectal temperature of 110°F, and realised too late that he had for four days been "incubating" Heatstroke. Energetic treatment during the next six hours, although it reduced the pyrexia unfortunately proved unavailing, and without regaining consciousness he died from respiratory failure. He was the first case of Heatstroke to be admitted that year, and the dramatic course of his illness stimulated the entire hospital staff to alertness in watching for such incidents.

The longer the prodromal period the worse is the outlook particularly in the gastric type of case. Many of the cases reported as occurring amongst patients under treatment in hospital for other conditions are /

are probably of this nature, and whilst no clinician can fail to appreciate the need for urgent treatment in the cases with an acute onset, it is probable that the peril in which these prodromal cases lie is not sufficiently well recognised. The recognition of this prodromal period is therefore of great importance, as steps can be taken to prevent the development of Hyperpyrexia which is always serious and often fatal. Any combination of the symptoms and signs described above should arouse suspicion and call for prompt preventive treatment. Especially helpful features are the bladder irritability, nausea and vomiting, absence of the knee jerks, any unusual mental changes, and the hot dry skin, though this latter is by no means invariable. Careful observation and occasional readings of the rectal temperature of suspect cases are advisable. In many cases, of course, the prodromal symptoms are so slight that the patient does not report sick, and remains at work until more serious symptoms compel him to seek medical advice, or perhaps until with the sudden onset of Hyperpyrexia and even of Coma he is brought to Hospital as a "stretcher case."

STAGE OF EXCITEMENT: The Prodromal period is succeeded usually by a short period called by Megaw[(150)] the Stage of Excitement, during which the temperature rises to 104° to 107°F. or more, and the symptoms of the Prodromal period are intensified. The patient is /

is restless; headache, frequently occipital, is severe and there may be tinnitus, photophobia, and diplopia. The skin is hot, dry and flushed and macular eruptions and petechial spots are recorded (97)(159)(169)(252). The pupils are equal, and are sometimes dilated, sometimes contracted; the knee jerks are sluggish or absent. The pulse is rapid and irregular, the respirations hurried and sometimes stertorous. The urine is scanty and contains albumin and sometimes casts.[168] Mental excitement is sometimes extreme, the patient babbling incoherently, and there may be wild delirium and even mania. According to Megaw [150] in their delirium the patients may rush about wildly and impulses to commit suicide especially by drowning are not infrequent. Heilman and Montgomery[97] say that extreme drunkenness may be simulated, which recalls an amusing incident of the 1932 heat wave in Allahabad. Two officers seeing a soldier stagger and fall whilst out for a run, brought him semi-comatose to hospital and with the aid of the nursing orderlies were about to carry out the appropriate "first aid" treatment for Heatstroke, the principles of which I had explained to all ranks. When I arrived I recognised the man as one whom I knew to have a remarkable capacity for alcohol for his age, which was 19. His temperature was normal, and the fact that his breath smelled strongly of alcohol prompted me to change the treatment, /

treatment, and, despite the reproachful glances of the "lay practitioners", a stomach tube was passed. Relieved of his stomach contents he revived, and claimed to have drunk 18 pints of beer between 4 and 6.30 p.m. which he had been endeavouring to "work off" when he collapsed.

STAGE OF COMA: Very soon, if not treated properly, the patient becomes comatose and the temperature rises still higher, being commonly in the neighbourhood of 110°F. The temperature is somewhat variable and may show rapid fluctuations, whilst for some unexplained reason it is sometimes subnormal.(251)(294) Unconsciousness is usually complete in this stage, though delirium sometimes persists. Fibrillary twitchings of muscles, and epileptiform or tetanic convulsions may occur, and Gauss and Meyer (294) observed that tonic and clonic spasma were sometimes focal, and involved often a single limb. The face is flushed and cyanosed, the conjunctivae congested, the skin often moist and clammy, and there may be incontinence of urine and faeces. The pulse is rapid, of small volume, thready and irregular, the breathing rapid or irregular, stertorous & often of Cheyne Stokes character. When semicomatose only the superficial reflexes may be lost, but when coma becomes complete all reflexes are often absent. The pupils which may be dilated in the earlier stages are now usually contracted except immediately before death /

death when with other sphincters they relax. They are equal, insensitive to light, and the corneal reflex is lost. If the patient is going to die, the respirations become slower, and more stertorous, the pulse weakens, and finally death takes place from asphyxia, after an illness lasting from a few hours to a day or so. The terminal temperature in Gauss and Meyer's[294] series varied from 95° to 110°F. and higher temperatures are common.

If recovery is to take place the temperature falls, as a rule rapidly, though complete defervescence may be delayed for some days. The respirations become quieter, the pulse rate slows, and the patient may fall asleep to awake much improved. On recovery from coma the patient is often irritable and disorientated, and may suffer from delusions and hallucinations. In Gauss and Meyer's[294] series the time taken for hyperpyrexial patients to return to normal varied from ten minutes to three days, and they found that when the return to normal took less than several hours, recurrences were usual. Frequently the fever lasts much longer, and there may be irregular low fever for eight to ten days or even for as long as three weeks,[159] with occasionally a rise of temperature to as much as 102° to 103°F. Relapses and recurrent attacks[403] are very apt to occur and the patient needs careful watching, since as Bonnyman[402] pointed out in 1864, he may fall asleep, and the disease be far advanced before/

before attention is directed to his state.

CLINICAL TYPES OF HEATSTROKE:

The clinical picture just described is that of the usual severe case of Heatstroke, but it does not cover all of the protean manifestations of this profound bodily disorder. The predominance in some cases of symptoms relating to the various systems has given rise to the differentiation of various clinical types.

Thus in the relatively uncommon but very fatal GASTROINTESTINAL or CHOLERAIC TYPE[159][252] there is sudden collapse with a temperature of 101° to 103°F, severe vomiting and profuse watery purging. In Gauss and Meyer's[294] series a few had rectal haemorrhages and vomited blood. The sunken eyes in a pale face, the pale clammy skin and cramps in the abdomen and legs point to the existence of dehydration and salt loss as in Cholera.

The CARDIAC TYPE Megaw[150] believes to be most commonly found in those with pre-existing heart lesions. The patient experiences a sudden pain in the cardiac region, the pulse is small and feeble, the rate 120 to 140, and death occurs from syncope. Marked cardiac dilatation and often a systolic murmur are common in severe cases of Heatstroke, and may persist after the attack. Cardiac dilatation was the only post mortem finding in a previously healthy young soldier who collapsed and died on a march on a hot summer/

summer day in England, various features of whose case made Heatstroke seem to be the most acceptable diagnosis.

The blood pressure in most cases of Heatstroke I found to be within normal limits, but Heilman and Montgomery[97] state that it is usually raised. During the production of Physical Hyperpyrexia Neymann (404)(405) noted a pronounced rise in pulse pressure, as the systolic pressure often rises, and the diastolic drops. These blood pressure changes he considered to have no clinical significance. Ferris[154] found a low blood pressure only in patients who appeared to be in extremis, and in none of his cases were there any signs of congestive heart failure. Most of the patients had a relatively high pulse pressure, and he believes that in typical Heatstroke the heart and peripheral circulation are entirely competent. Barclay[401] a military surgeon who in 1859 gave an extremely good account of the condition and of its treatment, considered it to be "entirely a nervous affection", and that the vascular system was only implicated secondarily. Electrocardiographic observations do not appear to have been made in Heatstroke, and in military practice at least, most of the cases occur where facilities for such examinations do not exist. In the pyrexia induced by physical means Vesell and Bierman[406] record various transient/

transient alterations in amplitude and intervals in electrocardiograms, but find no evidence of any harmful effect.

In the PULMONARY, RESPIRATORY, or ASPHYXIAL TYPE (150)(407) increasing cyanosis, dyspnoea, air hunger, or spasmodic breathing lead up to sudden prostration, with shallow or Cheyne Stokes respiration, and a feeble or imperceptible pulse. Bronchitis and Pulmonary congestion or Oedema occur and there are crepitant rales all over the lungs, and especially over the bases. Some such findings, especially basal congestion, are common in severe cases of Heatstroke, and Pulmonary Oedema is a terminal event in some fatal cases.

The Central Nervous System is particularly liable to damage from Hyperpyrexia, and few severe cases fail to show evidence of some such damage which is responsible too for many of the unfortunate sequelae of Heatstroke. Such terms as the COMATOSE, CEREBRAL, or PARALYTIC TYPES of Heatstroke (150)(225)(407) have been applied to cases in which the coma and convulsions already described are most prominent. The attack may come on suddenly with deep coma, recurring convulsions, vomiting, diarrhoea, and hyperpyrexia. The skin and dejecta have been said to have a peculiar "mousy" odour.(71)(407) If one wished to differentiate any special clinical condition as "Sunstroke" it is to this condition that one might perhaps/

perhaps apply the term since it sometimes seems to be due to intense regional overheating of the brain by the direct rays of a hot sun. In the less dangerous PSYCHOPATHIC or CONFUSIONAL TYPE[407] there is mental confusion, a muttering delirium, or excitement, and occasionally delusions, and suicidal impulses.

LABORATORY FINDINGS.

The laboratory findings in Heat Hyperpyrexia have already been described (page 87-92). Alterations of the acid-base balance are variable, alkalaemia being sometimes present whilst a terminal acidaemia and a rise of the non-protein nitrogen are common. Blood chlorides are sometimes raised and sometimes lowered. There may be leucocytosis, the differential count being usually normal, though occasionally there is a relative lymphocytosis.[294] The urine may contain albumin, red blood-corpuscles, pus cells, casts, indican, and acetone and diacetic acid, and the urinary chlorides may be diminished or absent. (20)(64)(71)(159)(252) The cerebrospinal fluid is normal but its pressure may be raised.[159] This rise of pressure is commonly seen when performing lumbar puncture for therapeutic reasons, but it does not seem to be marked, although I have had no opportunity of estimating it with a manometer.

COMPLICATIONS AND SEQUELAE.

It is important to bear in mind the possible co-existence/

co-existence of other diseases, especially Malaria which is likely to be endemic in places where Heatstroke is most liable to occur. Parotitis was present in three of Willcox's cases,[159] and intraocular effects such as neuroretinitis and choroiditis with subsequent detachment of the retina were described by Hotz[408]. Cardiac and respiratory complications, the latter especially apt to occur as terminal events, have already been mentioned. It is the damage to the Nervous system which is the gravest danger to those who survive the acute illness, and patients may be left with persistent nervous symptoms, the result of actual organic changes in the brain and cerebellum (71)(159)(252). Stewart[409] described a cerebellar syndrome following Heatstroke, and various nervous manifestations were attributed to "Sunstroke" amongst soldiers in the American War of the Rebellion.[410] These included various paraesthesias and pareses of upper and lower limbs, Hemiplegia, and Facial Paralysis, with impairment of sight and hearing on the right side and weakness of the right hand, sensation being normal. Messiter[411] also described a case of Hemiplegia following "Sunstroke," and Weissenburg[412] observed that numerous cases of motor lesions have been described, but that he was unable to find any instance of sensory disturbance. In a case of his own there were multiple nervous lesions producing/

producing acute cerebellar ataxia, loss of speech and spastic symptoms. Multiple neuritis associated with weakness and wasting of the legs, the Tibialis anterior being especially affected, defective articulation, nystagmus, squint, and diplopia are mentioned by Willcox.[159][252]

Mental changes are common, and headache, irritability, visual disturbances, loss of memory and powers of concentration, and even confusion and delusions, and convulsive seizures may persist for weeks or months. These symptoms are due as Megaw[150] observes to Cerebral Oedema which leads to a rise of intracranial pressure so that there is therefore usually bradycardia and persistent vomiting. These symptoms may clear up after a few weeks but there may be a progressive failure of brain power leading to insanity[168] Finlayson[413] observed that when insanity follows "Sunstroke" whilst mania may be the primary character, dementia usually soon follows, and Megaw[150] records dementia in about 10% of severe cases, and believes that most patients whose temperature has been over 108°F are left with an enfeebled brain, and mental changes like senility. Byam and Archibald[168] thought that some of these changes may result from chronic meningitis, which they found to be present on post mortem examination of such cases, and Wakefield and Hall[162] also note that/

that Heatstroke is often followed by Chronic Meningitis. Roch(274) recently described an acute benign meningitis due to "insolation" from which the patient recovered.

For some time after an attack of Heatstroke the patient may be unduly sensitive to heat, and after a very severe attack this increased susceptibility may be sufficiently marked to make further residence in a tropical climate inadvisable. There may for a time be increased susceptibility also to infections such as Pneumonia.

DIAGNOSIS.

The importance of early diagnosis cannot be exaggerated, for early reduction of the pyrexia lessens, not only the actual mortality of the disease, but also the likelihood of serious after effects. Recognition of the disease in the prodromal period, which would be ideal, is not of course always possible, as the patient may not come under observation until this stage has been succeeded by the stage of excitement or until he is in the advanced stage of coma. If the medical officer bears in mind the possibility of the occurrence of Heatstroke whenever the weather conditions are conducive to its production, any of the symptoms and signs described should arouse his suspicion, and he should not forget that Heatstroke may occur in a patient already febrile from some other cause. When the prodromal stage is past and the/

the patient is pyrexial, since delay may be fatal appropriate treatment should be begun whevener the diagnosis of Heatstroke seems probable, without waiting to exclude other possible causes of pyrexia which can be searched for after treatment has been instituted. It is extremely difficult and may sometimes be impossible to exclude Cerebral Malaria which may exhibit many of the features of Heatstroke such as hyperpyrexia, coma, delirium, convulsive seizures, and paralysis. Other forms of Malignant Malaria such as the algid, syncopal, gastric and choleraic, forms may resemble cardiac and gastrointestinal types of Heatstroke. Help may be obtained from the history, the presence of splenic enlargement, and from examination of the blood. The parasites of Malignant Tertian Malaria are notoriously hard to find, and the thick film method is useful in detecting scanty infections. The leucopenia and relative increase of large mononuclears found in Malaria are not observed during the actual paroxysm, when a leucocytosis may occur. Expert observers may derive helpful information from the finding of pigment in the leucocytes.

The exclusion of the virus diseases Dengue and Sandfly Fever may also be impossible in the early stages, but in neither case will the patient suffer from the application of treatment for Heatstroke, and the subsequent course should make the diagnosis clear.

It is not surprising that Cerebrospinal Fever with its early occipital headache, photophobia, irritability, and sometimes delirium and convulsions, is often mistaken for Heatstroke. The temperature is seldom so high as in Heatstroke, and there may be an initial rigor, whilst the correct diagnosis may be suggested by the presence of head retraction, Kernig's or Brudzinski's signs, strabismus, leucocytosis, and herpes, though this is often late in appearance. The Knee jerks are increased, not absent as in Heatstroke, and Babinski's sign is positive in about 10% of cases. [233] Lumbar puncture should be performed in doubtful cases.

In Cerebral Haemorrhage the pupils may be unequal and there is conjugate deviation and some paralysis. When pyrexia is associated with Cerebral Haemorrhage, as it is especially in Pontine lesions, the fever follows the onset of coma instead of preceding it as in Heatstroke.

From coma due to such causes as uraemia, diabetes, alcohol, or opium and other narcotic poisons Heatstroke may be distinguished by the high temperature. Examination of the fundus oculi, the blood pressure and the urine would of course be carried out if uraemic or diabetic coma were suspected. Alcoholic coma is seldom complete, the pupils are dilated, the breathing may be deep but not stertorous, and the/

the breath smells of alcohol. In opium poisoning the pinpoint pupils and the fact that the coma is gradual in onset are additional points of difference.

Convulsive forms of Heatstroke must be distinguished from Epilepsy, and other conditions associated with epileptiform attacks, in which connection no army medical officer is likely to forget Cysticercosis which has attracted so much attention since in 1934 MacArthur(414) stressed its importance as a cause of epileptiform fits and other nervous manifestations especially in those who have served abroad. (415) to (422).

PROGNOSIS.

The prognosis is always serious and the patient must be considered to be in grave danger during the first twenty-four hours, except in mild cases when the temperature has been normal for some time and the general condition is satisfactory. The patient is usually out of danger in a day or two, but during the pyrexial period which often succeeds the acute attack one must, as we have seen, watch carefully for the occurrence of relapses, although whilst the temperature remains below 102°F. there is probably no grave cause for alarm. The average mortality varies according to Castellani and Chalmers(169) from 15 to 25% but may be as high as 51%, and Megaw(150) estimating it at 25 to 40%, points out that/

that such figures are of little value on account of the great variations in severity. He observes that the prognosis depends on the height reached by the temperature before it is controlled by treatment. In one series when this was 107°F. the mortality was 8·3%, when 107° to 109oF. 29·2%, and when over 109°F. it was 69·2%. Rogers(150) found that patients who had been unconscious for over three hours before being treated did not recover, whilst if the duration of unconsciousness had been less than one and a half hours, they all recovered, and of those in intermediate categories some recovered and some died. Alcoholism, debility, old age, obesity, co-existing diseases, or pre-existing heart, lung, or kidney diseases adversely affect the prognosis, as do the occurrence of cyanosis or convulsions. Great importance is attached by Willcox(159) to the knee jerk. Whilst the knee jerk is absent the prognosis is bad, its return is a good omen, and whilst the knee jerk continues to be absent after recovery from the acute condition there is risk of a recurrence. Byam and Archibald(165) find that recovery generally takes place when the blood creatinin is under 4 mg. per 100 v.v. and Gradwohl and Schisler(423) found that a high non-protein nitrogen pointed to a severe case. Some cases with high non-protein nitrogen but few symptoms were fatal, whilst others with severe symptoms but low retention recovered.

McIntosh/

McIntosh[424] found that an invariable sign of recovery in unconscious cases was vomiting.

It may be said that with early diagnosis and prompt treatment, death should rarely occur but that the prognosis for complete recovery depends upon the duration and height of the fever, and upon the age, previous health, and habits of the patient.

TREATMENT.

When the prevailing climatic conditions are such that cases of Heatstroke are to be expected special provision should be made in Hospitals for their reception and treatment. A ward artificially cooled by air conditioning apparatus such as that described by Rennie,[425] and Anderson and Sproule[426] would be ideal but is not generally available in our military hospitals. A ward on the ground floor should be selected and kept cool by such methods as the use of kuskus tatties which will be described later. Electric fans are of course provided in all military hospitals in the Plains. Morton[160] improvised a refrigerating plant by filling the radiator of a Leyland lorry with ice and blowing air through it with a fan. Dodd and Wilkinson[427] prevented effects of heat in children in hospital by means of three electric fans in the ward one of which blew upon a tub of ice and salt whilst the other two blowing air against the ceiling, kept the air in the ward in constant motion. Patients can be provided with/

with individual cool air chambers by covering cradles with sheets kept moist, and fanned with electric fans or with ice in waterproof bags suspended inside them and a table fan blowing the cooled air through them (148)(160). For the reduction of pyrexia in acute cases it is convenient to have adjoining the ward a special room with a stone floor well drained if possible, with electric table and ceiling fans, and water laid on, with some arrangement for spraying the patient.

The only furniture needed is a "charpoy" or Indian bed with a wooden frame to which a string mattress is attached. Patients from the wards will be brought here wrapped in wet sheets, but pegs may be provided for the clothing of those admitted direct to this treatment room. All that is needed for venesection, intravenous infusion, and lumbar puncture, should be kept in readiness in the ward, and the orderlies should be instructed in the nursing and observation of Heatstroke cases, the salient points in which may well be outlined on a typed notice hanging in their "duty room."

Reduction of the temperature is the first and most urgent indication in the treatment of Heat Hyperpyrexia, since the patient's chance of recovery and of avoiding serious sequelae largely depends upon the duration of the pyrexia. By far the best way of reducing the temperature is to make use of Nature's method/

method of the evaporation of water from the skin. The patient, stripped and laid on a string mattress to allow of free circulation of air round the body, is sprayed with water, and vigorously fanned with electric fans, hand fans or towels. Whilst this is being done a close watch is kept upon the rectal temperature and when this reaches 102°F. or so the procedure is stopped, and the patient, wrapped in blankets, is put to bed with hot bottles applied to the limbs and trunk. This encourages sweating, and avoids the risk of too rapid a fall in temperature to below normal, which sometimes occurs and may result in dangerous collapse. The foot of the bed may be raised. The patient must be carefully observed for any recurrence of the hyperpyrexia which would call for a repetition of the procedure. Such recurrences may occur very suddenly, and it was Gauss & Meyer's (294) experience that they/are harder to treat than the initial pyrexia. It is not necessary to use iced water for the spray, as the difference in calories lost by evaporation is small between warm and iced water.(204) Rubbing of the skin with ice, which is often practised, and the use of ice packs are to be deprecated as being not only less efficient in reducing pyrexia but also potentially dangerous. Hill (159)(204) found that the evaporation of water at body temperature takes away 0·59 calories per gramme whereas the melting of ice takes away only 0.08 calories/

ories per gramme. If the skin is rubbed with ice the cutaneous capillaries are constricted, and the blood is driven away from the surface where it is needed for heat loss to the deeper parts of the body. Apart from the interference with heat loss Heilman & Montgomery[97] believe that this sudden change in circulation may strain the Heart and cause collapse. The application of ice bags or blocks of ice to the head and neck and inguinal regions may however be helpful. (71)(150)(252)(294). Sponging with water containing alcohol has been recommended but does not seem to have any advantage over the method outlined above.[294][428] Army medical officers are well aware of the value of the method of reducing pyrexia which I have described, and indeed Pembrey[429] stated that he thought that the credit for the introduction of this method seemed to be due almost entirely to the Medical Services of the Army. Darrach[430], who in 1859 advised rubbing of the body with ice, commented upon the success of Beatson a British army surgeon with affusions of cold water to the chest and epigastrium. When working with some Assistant Surgeons of the Indian Medical Department - men who give invaluable assistance to the Army medical officer in India, and from whom helpful hints on tropical medicine can often be picked up by a young medical officer who is not too proud to learn from subordinates/

nates - I have found that they are apt to retain a lingering reverence for rubbings with ice. This attitude is perhaps hardly to be wondered at since this method has certainly been practised for a long time, and is still included amongst the treatment recommended by some text books. (233)(302)(431). It may therefore be advisable in order to avoid lack of uniformity in the campaign against Heatstroke to ascertain one's Assistant Surgeon's views on treatment, and, if necessary, tactfully to make him a convert to the evaporation of water method.

If the relative humidity is too high for full advantage to be taken of evaporation, or if no fans are available, the patient may be immersed in a bath of iced water, the rectal temperature being observed as before. Whilst he is in the bath vigorous friction of the skin must be kept up, to keep the skin capillaries filled with blood and so to aid the elimination of heat. This method of reduction of temperature was the one most favoured by Ferris[154] in his recent study of the subject, and is certainly a valuable one when for any reason evaporation of water from the skin cannot be employed.

Such immersion in cold water may help to stave off a further rise of temperature in cases who in an emergency cannot be immediately attended to, and Woodyatt[432] describes how in an extensive outbreak of/

of Heatstroke during operations on the Indian Frontier the lives of many were saved by being immersed in a large tank whilst awaiting evacuation. The use of iced water enemata is often recommended, and Rogers (433) found them to be useful.

According to Hill[55] however, 70 grammes of water evaporated from the skin remove as much heat as 1000 grammes of iced water used as an enema. The iced enema has moreover the serious disadvantage that it deprives one of the most valuable means of observing the temperature of these cases. It has however a place in treatment when one is coping with a great many cases, as an orderly can administer enemata to cases with whom one is unable to deal immediately.

Antipyretic drugs are to be avoided as they may be dangerous, owing to their depressant action on the Heart, and are in any case of little or no use in reducing the temperature. As Barbour[434][435] pointed out such drugs as aspirin do not stimulate a depressed heat regulating mechanism, the normal individual responding to them by an increase in carbon dioxide output and heat production.

In any case where there is a suspicion of Malaria a blood film will have been taken, but a history of Malignant Tertian Malaria or an enlarged spleen call for the intravenous administration of 6 to 10 grains of Quinine dehydrochloride without waiting for confirmation by the finding of plasmodia.

Whilst the temperature is kept within reasonable limits by the treatment just described one must endeavour to determine if any other concomitant pathological effects of heat are present requiring treatment. There will often be varying degrees of dehydration and salt loss. Thirst is a symptom of Heatstroke, and conscious patients should be encouraged to drink copiously, and lemonade, barley water, or weak saline solution may be given. Fluids should be pushed by the mouth and by other routes until free Kidney action is evidenced by a distending Bladder. Fluid is especially important in cases with persistent vomiting and in those which had an insidious onset.

We have seen that the giving of Saline to dehydrated patients is not without its drawbacks as it may cause a tendency to acidosis and even to an increase of the dehydration, and that the use of saline solutions containing lactates, or glucose has been recommended (pp.156-157). Another possible contraindication to intravenous saline in Heatstroke is in cases of incipient respiratory failure in which the development of Pulmonary oedema is to be feared, since it has been found that intravenous saline may cause oedema.[(436) to (438)] Maddock and Coller[(436)(437)] found that this danger could be avoided by giving 5% dextrose in distilled water intravenously, as this provides for a normal water exchange. Cramer[(439)] considered/

considered that intravenous saline was contraindicated in cases of Heatstroke, and Willcox[159] found that it caused recurrence of convulsions in convulsive cases. Many authorities however recommend the giving of saline in Heatstroke.[150][164][397][407] Hill[55] found saline helpful, and gave gum saline to collapsed cases, and Willcox[159] and Manson Bahr[71] recommended it for choleraic cases. McMillan[298] found that frequent injections of small amounts of isotonic glucose and saline into the rectum gave good results. Professor Lambie of Sydney University tells me that he gave hypertonic saline by the mouth and intravenously to Heatstroke cases in Mesopotamia during the War. I found that intravenous infusion of saline, preceded in plethoric cases by blood-letting, benefit~~t~~ed severe cases. The occurrence of cramps is an obvious indication for saline administration.

Useful although the giving of saline may be, it should again be emphasised that the reduction of pyrexia must come first, as an unfortunate experiment of Ferris' indicates.[154] Since undue weight had in America been attached to the replacement of salt loss, and a misconception had, he thought, developed that the problem of Heatstroke was one purely of replacement of sodium chloride lost in sweating, he refrained from giving the routine treatment for reduction of pyrexia in one case, giving only large amounts of normal saline intravenously.

The/

The patient's temperature remained elevated, he remained in coma, and died in a few hours.

The administration of alkaline solutions by the bowel or intravenously is often advised, and the intra:venous infusion of one to two pints of a 2% solution of sodium bicarbonate has been recommended(82)(164)(168)(302)(407). Willoughby and Aslett(440) used an alkaline solution advised by Rogers (150) consisting of Sodium chloride 90 grams Calcium Chloride 4 grams Sodium bicarbonate 160 grams and Water to one Pint. This solution Rogers gives at a temperature of 60°F. and it can be given intravenously or per rectum. Willcox[(159)252)] found that rectal injections containing two drachms of Sodium bicarbonate to a pint were beneficial, and both he and Manson Bahr[(71)] advise the administration of 30 grains of Sodium bicarbonate every three hours to gastric cases. Morton (160) found the use of intravenous alkalies and glucose disappointing, and Hill, [(55)] believing that there is usually no acidosis, holds that there is no need for sodium bicarbonate, which is moreover contraindicated in cases with quick shallow respiration, in which alkalosis may be present. As already described (pp. 88-91) the behaviour of the acid-base balance in Heat Hyperpyrexia is variable, and in the treatment of such urgent cases time does not allow of the laboratory investigations necessary to confirm the presence of a suspected acidaemia. In a gravely ill case/

case air hunger and perhaps acetone bodies in the urine might lead one to suspect a terminal acidaemia but one must remember that the presence of ketone bodies does not necessarily indicate acidaemia, whilst an acid urine is not incompatible with the presence of alkalaemia. Probably in most cases alkalies are not likely to do good, although they may not actually do harm, since the body is more tolerant of a reaction more alkaline than of one more acid than normal, and if severe alkalaemia were being caused one might observe the occurrence of tetany as a warning. Marsh[82] suggests that the good results reported from the use of alkalies may be due to the conversion of sodium bicarbonate in the body to sodium chloride.

Measures directed to preventing Respiratory and Cardiac failure have an important place in treatment.

Venesection with the removal of ten to twenty ounces of blood is of great value in asphyxial and plethoric cases (55)(159)(252)(298)(397)(407)(441). Its good effects are often striking and, as it affords relief to the labouring right side of the heart there is no reason for deferring it, as Heilman and Montgomery[97] suggest, until symptoms and signs of Pulmonary oedema occur. Willcox[159] found it superior to sedatives in the treatment of cases with violent delirium and convulsions. Artificial respiration may be required to combat respiratory failure, and should be kept up whilst there is hope of resuscitation./

tion. Rhythmic traction on the tongue has been recommended,[150][397] and Willcox[159] gave oxygen.

Stimulants may be necessary and those most often used are camphor, strophanthus, adrenalin, and the caffeine group of drugs. On account of the tendency to petecheal haemorrhages in the brain, meninges, heart and elsewhere it has been suggested that these stimulants should be avoided if possible, and used only as life-saving measures.[97][397] Aromatic spirits of ammonia by the mouth, by intramuscular injection, or by inhalation, is recommended by Fantus. (397). Although strychnine is employed by some authorities (252)(294)(431)(442) it is generally held to be contraindicated by its tendency to encourage the convulsions which may occur in Heatstroke(71) (164)(168)(298)(407). When relief of intracranial pressure is indicated, lumbar puncture with removal of 20 to 40 c.c. of cerebrospinal fluid will be found to be most beneficial. If for any reason it cannot be performed the intravenous injection of 100 c.c. of 50% glucose or 70 c.c. of 15% saline solution is helpful, or six ounces of a 50% solution of magnesium sulphate may be given by the rectum.

Restlessness, delirium, and convulsions, may call for the use of sedatives, although as mentioned above, Willcox found venesection to be more efficacious in relieving such symptoms. Bromides and chloral, which may be given by the rectum, morphia and/

and hyoscine hypodermically, are the sedatives most used, and convulsions may be treated by the cautious administration of chloroform.[252][407]

Cramer[439] found chloral to be the most suitable drug, but from an analysis of the cases treated in the army in India in 1932 McMillan[298] concluded that it should be avoided. I found that a drachm of chloral hydrate in a rectal enema, as advised in the official memorandum compiled by MacArthur,[407] was very useful in some delirious cases.

It will be seen that whilst the patient is gravely ill, the medical officer, especially if he has several cases to attend to, will find his time fully occupied, and as the cases in the early stages show so marked a tendency to relapse, he will not be able to be for long away from the hospital. Any privations which he may have to undergo will be amply compensated for by the knowledge that this condition is one in which the general practitioner or physician is privileged to engage in a hand to hand struggle with the Angel of Death, appearing in the heroic rōle for which his surgical colleague is more often cast.

Patients who have passed the acute stage should be treated somewhat like cases of concussion, being kept strictly at rest in a cool ward, and forbidden to read if there has been any suspicion of mental impairment. The diet should be light, consisting largely of milk, vegetables, fish, and eggs. Bland fluids/

fluids should be taken freely, and constipation should be avoided. Those cases in whom there is a persistence of a low grade pyrexia should be especially carefully watched. I found that they seem to be benefit~~t~~ed by small doses of calomel with salol. As soon as convalescence is well established a change to a cool climate is desirable, and all excitement and fatigue should be avoided for several weeks. Alcohol should be forbidden, and abstinence for a long time after recovery should be urged. The convalescents should be kept in the Hills for at least three months during which time they should be kept under medical supervision.

If cerebral manifestations have been prominent, or if headaches are persistent, "brain work" and work in offices should be avoided. For cases in which severe headaches or convulsions persist for as long as three months Rawling[443] advises subtemporal decompression, as these symptoms may be due to cerebral oedema resulting from temporary or permanent impairment of the tunica intima of the cerebral sinuses and veins with collection of an excessive amount of cerebro-spinal fluid.

If the attack has been a severe one, and the patient has been left with serious after effects or is unduly sensitive to heat, the question of invaliding him out of the country and even of his discharge from the service may obviously have to be considered, and it is probably advisable that a man who has had two attacks should not be allowed again to serve in tropical countries.

PREVENTION OF EFFECTS OF HEAT.

EDUCATION ETC.

The first step in the campaign for the prevention of Heatstroke may well be taken before the potential victim leaves the temperate climate to which he is accustomed, by means of lectures to troops on life in hot climates, and the avoidance of effects of heat. In the lectures on the preservation of health in general, which are given every year to all ranks, I have always included a few remarks on this subject and lectures should be given on board troopships bound for the East. The soldier's family should not be forgotten, and provision is made for them by the issue of pamphlets on the subject similar to those which exist for the instruction of officers and other ranks on the preservation of health in hot climates, precautions to be observed when travelling by rail, and such matters.

A thorough physical examination is always carried out, although it must be admitted that it does not at present include the test meal thought essential by Sir Arthur Hurst, (see p.149). The period of the voyage affords a good opportunity, of which advantage should/

should be taken, for men to become gradually sun-tanned, and so to acquire the pigment protection which will enable them to expose their bodies freely for heat loss without suffering from the injurious action of the sun on their skin.

BARRACKS, ETC.

The provision of suitable Barracks, Hospitals, Trains, and Troopships is important and in this respect the soldier is in general well catered for.

Houses should be thick walled so that they may heat up slowly by day and cool by night. They should have double roofs, and good wide verandahs to prevent the sun beating in at the windows, and they are usually white or yellowish in colour, since white surfaces absorb least heat. If there is any chance of radiant heat from sand or ground glare reaching the inside walls of a room, the walls and particularly the ceiling should be so painted as to be good absorbers of heat and not reflectors, and may be distempered in dark green or some such colour. For dry heat lofty rooms with doors and windows which can be properly closed in the heat of the day are preferable, and clerestory windows are useful, as they allow heated air to escape at night. During the day the house is kept shut and electric fans are used, and the house is opened up in the cool of the evening. For a moist climate good through ventilation is essential/

essential. Most barracks are now mosquito proofed, and it is not too much to hope for that, in the near future, the hospitals at least and possibly other buildings will, in the hotter stations, be provided with refrigerating plants. Meanwhile quite a lot can be done to make existing buildings cool on the lines mentioned when dealing with heatstroke wards (p.189) When, in the hot weather, a large proportion of the troops are in "cold storage" in the Hills, which is a valuable prophylactic measure, those remaining in the Plains should as far as possible occupy ground-floor barrack rooms, as upper-storey rooms have been found to produce a greater number of Heatstroke cases.[225] The use of kuskus tatties is especially helpful when the temperature is high and the humidity low. The tattie consists of a wooden framework covered with a thick matting of brushwood or the fibre of kuskus grass, which is kept constantly moist with water either by hand or by some artificial sprinkling device. They are placed over the doors on the windward side of the bungalow. I have heard them objected to on the grounds that they increase the humidity of the room, but it will be remembered that Yaglou[209] recommends artificial humidification in order to lower the effective temperature in hot dry rooms, and Megaw[150] believes that the cooling effect of the evaporation of water has not been adequately exploited/

exploited in dry hot climates. Certainly the effect of the kuskus tattie is to produce a most pleasant coolness. The efficiency of the tatties may be increased if they are installed in the manner suggested by Megaw[150] of which the cost is apparently not much greater than the usual method of merely placing them over the door frames. A good electric exhaust fan is fitted into a hole in the door, and to the outside of the door is fitted a frame slightly smaller than the door and about eighteen inches in depth. To this frame the kuskus screen is attached in such a way that all the air sucked into the room must pass over it. The tattie is kept constantly moist by drops from a cistern placed above the door in a shaded place. If a long narrow room is selected and a second exhaust fan is fitted at the other end, all doors and window being tightly closed, a constant flow of cool fresh air is provided. Dust and insects are excluded by such fittings. If a barrack room or a ward is built on this principle there is a saving in cost, as the height of the room is less and the width too can be reduced. This system is not effective if the outside air is damp but then the heat is less intense, and the ordinary electric fans suffice. In many cases the flow of air created by the two exhaust fans is enough, if the flow of water over the kuskus screen is cut off. In places with a cold winter and a hot summer a coil of/

of hot water pipes can be used instead of the kuskus screen in cold weather.

The researches of Crowden[444] on insulation against heat and cold have opened up great possibilities of increasing the comfort of those who live in the Tropics. The principles involved are the fact that bright metallic surfaces radiate very little heat compared to other surfaces, and reflect much of the heat which they receive; the low heat conductivity of air; and the fact that an air-proof material hinders the passage of heat by convection currents. The insulator used consists of a sheet of material covered on both sides with aluminium foil, so fixed as to divide the air space across which the transference of heat is to be hindered. Huts of which the ceiling and walls were lined with three-ply wood or asbestos beneath which was a layer of reinforced aluminium foil with an air space of half an inch on each side of it, (or, to put it differently, an air-space of one inch with a layer of reinforced aluminium foil in its centre) were substantially cooler than those lined merely with asbestos or three-ply wood two inches below the corrugated iron roof.

During manoeuvres or active operations troops must live in tents and these must be of the double-fly pattern. Damping the outer layer helps to cool the tent by evaporation, whilst whitewashing or lime-washing the fabric inside and outside markedly lessens solar absorption[444a] and Crowden's method of insulation/

sulation is also valuable, although there are certain drawbacks to be overcome, such as the risk of damage, by folding, of the aluminium foil insulator which divides the air space between the two layers of canvas.

In trains provided with sunblinds and fans it is possible to keep fairly cool, and damping of the floors, and the presence of a block of ice in the compartment help in hot weather. One should avoid the excessive enthusiasm of a young officer who on leaving for India had been, as one usually is, overburdened with advice from old campaigners on "Keeping fit." He had been instructed to get two seers (about 4 lbs) of ice put into his train compartment, and on reaching Karachi he enquired the price of ice and was told so much per maund. Thinking that this must be the half-remembered word used by his advisers he ordered two maunds, (about 160 lbs) and with some difficulty clambering over the icebergs into a top berth he spent a wretched night, and arrived at his destination some days later with Pneumonia.

Megaw[(150)] thinks that blocks of ice do little to mitigate the heat as the air cooled by them falls to the floor, and is swept through the chinks of the door, and he thinks keeping the floor of the carriage constantly wet is sufficient. He finds a wet towel over the body with a fan playing upon it very/

very helpful, and to avoid chilling the towel can be placed over an improvised surgical cradle. Kuskus tatties can be used in Railway carriages and the movement of the train acts as a substitute for the exhaust fans. Crowden's[444] insulator can also be fitted, and he advises the use of dark green distemper on the inside roof of compartments, instead of white, to lessen the reflection of heat on to the passengers. When troops are to undertake a long rail journey, an abundance of cold drinking water must be provided, and officers should carefully inspect the train before starting, and at frequent intervals, to ensure that conditions are comfortable, and all fans in working order, and that there are no men in need of medical care.

On Troopships, although the soldier naturally cannot enjoy quite so much space as in his barracks, overcrowding and unsatisfactory conditions are prevented by various regulations governing the use of this form of transport. Good ventilation of the troopdecks is ensured by fresh air blowers, wind sails, and scoops, electric fans and Thermotank ventilation. The use of sunhelmets is enforced, and open deck spaces are covered with double canvas awnings when in hot climates. A plentiful supply of drinking water, mineral water, and ice is provided and the medical officer has power to increase the scale on/

on which these are issued. If any criticism is justified it is that it is occasionally impossible to obtain sufficient space for all ranks to sleep on deck in the open air, which is such a relief when the Red Sea is reached, despite the unfortunate necessity of being awakened in the early morning by showers of water when the decks are swabbed down.

Crowden(444) has applied his insulating method to steamships and Thompson(445) found that if it is used to insulate such hot structures as steam pipes, hot water and cooking appliances, and the funnel casing, great relief can be afforded.

Wherever troops are stationed or encamped at any distance from a hospital a centre for the first-aid treatment of Heatstroke should be provided, and one may with advantage be maintained at large railway junctions or at large stations in desert country.

The more permanent first-aid posts may be constructed with Crowden's aluminium foil insulation, a double roof projecting to form a verandah and a drain:ed stone floor like that of the washing room of a garage. They should be situated if possible in a place where winds are available. One or two charpoys with a fan over each will be required, and a tank containing water, ice-cooled perhaps to about 50° or 60°F., with hose and sprinklers. A plentiful supply of drinking water and of saline solution should/

should be provided and such equipment as one or two thermometers, an enema syringe, some vaseline, a hypodermic syringe, stimulants, including sal volatile, sheets and blankets and stretchers, ice bags and an ice box for storing ice and cooling drinks. Temporary or improvised stations will have double roofs which may be improvised by fixing rushwork matting a few inches above the roof, and perhaps kuskus tatties. Similar equipment will be necessary, the post may well be located by a natural source of water, and drinking water may be stored in earthenware chatties, which keep it cool, and in place of electric fans hand fans will be necessary.

CLOTHING

Clothing material for tropical wear should be light and permeable to air, yet capable of excluding the direct rays of the sun which it should absorb as little as possible. White is of course the colour which absorbs least heat, and black the most, and of intermediate colours pale blue and Khaki are good. The clothing should not constrict the body anywhere, especially at the neck and waist. Free ventilation and good thermal conductivity are important, and the material should not be liable to be injured by sweat nor by repeated washing. No material exists which will fulfil every requirement, being cool and yet preventing chilling when the temperature falls. Woollen/

Woollen materials have good evaporative powers and their elasticity prevents clinging, but they are too thick and prevent heat radiating from the body, they have a low conductivity, and they imbibe much moisture in which process heat is generated. Also they shrink, and become hard with repeated washing, and by irritating the skin they predispose to Prickly Heat. Linen, cotton ~~or~~ + silk have a high conductivity, and are much more suitable, provided they are loose enough to allow for free ventilation and to prevent them clinging to the skin which would cause them to retain heat. A loosely woven material is in many respects the best and gives good ventilation, but it also allows penetration by the sun's rays from which blistering of the skin may result if a good tan has not been acquired. As Hill[177] says, close-meshed clothes of fair thickness are found more comfortable when there is much sun and a high evaporative power. The light, khaki, closely-woven drill of which our tropical uniform is made is quite satisfactory, although it is perhaps a pity that in the interests of smartness, a certain amount of starch is usually added when the uniform is washed and pressed. However, when the weather is hot the uniform jacket is rarely worn and all work is done in open necked short sleeved shirts, and shorts. The shirts are now mostly of a thin, closely-woven, khaki cotten material, and the shorts of the khaki drill, fixed/

fixed by buckles at the waist. To avoid constriction braces would be better but are less smart, although in at least one regiment broad white canvas braces are worn and give quite a smart appearance. Few who have not seen it can realise how smart the soldier looks in shorts, and when one returns to the, at first rather irksome, confinement of trousers one is inclined to wish that soldiers and others would adopt this excellent dress in temperate climates also. After sundown trousers should be worn to protect the legs from mosquitoes and if the evenings are chilly something warm round the abdomen may be advisable as a precaution against chills. When jackets are worn the neck should be left open to provide a "flue" for ventilation, a practice advised by a committee in 1909[464] which might be more generally followed by regiments serving at home in hot weather. The kilt, excellent though it is for temperate climates is not good in the heat, especially those worn by the rank and file of which the waist part is lined with woollen serge which after a march is sodden with sweat, and to avoid chilling has to be kept on for an hour after reaching camp, being sometimes turned inside out to facilitate drying. In any case, dangerous though it may be to breathe such heresy north of the Border, this becoming and comfortable dress is highly unsuitable for modern warfare. A valuable recruiting agent in peace, in war/

war it exposes its wearer to added peril from mustard gas vapour, and does not take kindly to periodic "delousing". If one is to suppose that, even should the impregnation of clothing against mustard gas penetration be regarded as practicable, the Highland soldier like all the other troops, officers and men, will have to be clad in some universal uniform which can be frequently replaced at decontamination centres, and when one realises the insuperable obstacle to blowing the pipes imposed by the wearing of a respirator one has to admit that war will be stripped of its last vestige of glamour.

The vest and drawers are usually of thin cotton, and the only undesirable feature is that the latter usually have elastic waist bands which interfere with the free circulation of air. Cotton cellular material would be better but is more expensive and wears out more quickly from sweat and frequent washing. A useful provision for ventilation is to have the shirt split beneath the armpits.

The wearing of a spine pad some nine inches wide, made of thick, air-containing material is sometimes recommended as it shields the spine from the sun, covering it with a mass of material which is slow to warm up.(159)(177) Personally I do not advise one as it prevents the evaporation of sweat, and the part of the shirt which it covers soon becomes wet and sodden, and the spine pad itself as

Hamilton/

Hamilton[225] says comes to resemble a poultice. If used the spine-pad should always be outside the straps of any equipment, or braces which are worn.

Two types of military sun helmet are in use - the Wolseley service topi made of cork and the thick pith helmet, whilst Indian troops wear pagris of various patterns. The Wolseley topi stands up better to hard usage and to rain, but the pith helmet is lighter and cooler, and being quite cheap is often brought into use during the hot months, a practice which is to be commended. These helmets are of course so constructed as to allow free circulation of air round the head, the head band being held away from the side of the helmet by short struts. It has already been explained that special coloured linings in the topi are of no benefit, and are in fact the reverse (p.108). Crowden[444] found that sticking aluminium foil to the inside of the helmet reduced the heat radiated to the head to 1/10th or 1/12th of what it was before, and this inexpensive improvement should soon be universal. If a second or drop-lining of aluminium foil attached to both sides of a sheet of fabric is fitted, use is also made of the high heat-reflecting properties of the metallic surface, and the layer of air between the drop lining and the helmet acts as a poor conductor of heat. Iwabuchi[447] found that helmets painted with/

with aluminium powder or zinc oxide were much more efficient than unpainted ones. Onogi[(448)] records that military steel helmets lined with cork are much more comfortable and the temperature inside them 5·7°C lower than in unlined ones, whilst lining them with flannel reduces the temperature by 3.4°C.

The usefulness of umbrellas and handfans advised by Willcox[(159)] cannot be doubted but their employment by the military population can hardly be recommended, although Sir Charles Napier the conqueror of Sind used a large umbrella, even when mounted during very hot weather. The soldiers' families might well be advised to use umbrellas and hand fans.

The ill effects of glaring tropical light on the eyes have been mentioned (p.141) as well as its possible general effect on the vaso-motor mechanism (p. 123). The relief afforded by wearing tinted glasses is immense (159)(177)(244)(291) and I suggested in 1932 that they should be issued to all troops. They are quite cheap and men might be encouraged to buy them for themselves.

DIET ETC.

In the selection of a suitable diet for the soldier in India one must make due allowance for our racial tendency to avoid any attempt to conform to the habits of any foreign country in which we may be living. The Spanish proverb about mad dogs and Englishmen is too well worn to need repetition, and Andre/

André Maurois[449] recently observed that whilst the French colonist attempts to adapt himself to the customs of the country, the Englishman must have on the slopes of the Himalaya or on the borders of the Soudan his club and his tennis. Indeed to the "man in the street" that absurd figure the "pakka sahib" dining in the jungle in a stiff shirt and dinner jacket is probably the type which has "made our Empire what it is." I have lived in one mess in India where the midday meal was hardly distinguishable from what one would have during winter at home, whilst on the two half holidays even in the hottest weather there was usually a ponderous mixed grill, the mere contemplation of which was enough to bring beads of sweat to the brow. In another mess, lunch consisted always of egg or vegetable dishes and various fruits, and we all found this very much better than meat. The private soldier is, in matters of diet, still more rigidly conservative, and has a wide range of likes and dislikes, the latter strangely enough often including mutton and fish. Any alterations in his diet which he suspected of being directed by economy he would resist, and he has of course a voice in these matters which he is encouraged to use on messing committees. When the slogan "Eat a herring a day and save the fishing industry" was being used I tried to introduce fresh herrings, which are almost a staple article of diet in many parts of the Highlands/

Highlands, into the breakfast menu in a certain barracks on the Scottish coast, but without success. The quartermaster from his long experience of soldiers assured me that they would not appreciate herrings whilst they knew they were cheap,

Eijkman[263] observes that tropical aborigines live largely on vegetables, taking a diet rich in carbohydrate and poor in protein and often in fat. In Batavia 25% of the protein intake of Europeans was from vegetable food whereas in the case of Malayans the figure was 70 to 80%. Benjamin Rush[450] in 1777 in his "Directions for Preserving the Health of Soldiers" picturesquely emphasised his recommendation that the diet of the American soldier should be mainly vegetable by saying "If every tree on the Continent of America produced the Jesuit's bark it would not be sufficient to preserve or to restore the health of soldiers who eat one or two pounds of flesh a day." There is much to be said in favour of conforming to the habits of the natives by reducing meat and eating mainly vegetables and fruit in hot weather[451][452] and Sir Patrick Manson[453] thought that Europeans in the Tropics often take too much protein and do not modify their diet enough, or make sufficient use of local vegetables and other native material. Hill[177] suggests as suitable foods for hot weather, fruits, salads, green vegetables, boiled rice, a little fish or bird and dairy produce, but remarks that the reduction/

duction in protein must not be carried too far. The races in India which eat animal protein are, he says, the most virile, and Aykroyd[(454)(455)] recently emphasised the extensive malnutrition amongst Indian children due to their vegetable diet. It was pointed out (page 126) that in circumstances likely to lead to Heatstroke carbohydrates are a better source of energy than fats or proteins, as they cause less heat production, but that at the same time food requirements for similar work are about the same in hot as in temperate climates. Drinker[(251)] finds that there is no logical reason for a highly restricted protein intake and the necessity for giving muscular workers an adequate supply of protein cannot be doubted. The standard ration issued in India has a somewhat higher caloric value than that issued in the United Kingdom, and actually includes more meat, possibly because the meat, and especially the mutton, obtainable in India is sometimes of a poorer quality, There is however no reason why the soldier should be given his meat during the heat of the day, when it must add an additional burden to his heat-regulating mechanism. I used to urge commanding officers to give their men a light midday meal, and to make the main meal an evening one, but this was often not practicable. Nowadays, with the greater facilities for giving men supper, there should be no difficulty in making this rational adjustment in the daily menu.

Since/

Since it is very likely that minor degrees of hypochloraemia may be responsible for much ill health in the Tropics and may predispose to Heatstroke (pp. 66-81), the diet should contain a liberal supply of salt and when conditions are especially severe the routine administration of some form of saline solution would probably be beneficial.

Brodkin(104) and Van Zwalenburg(105) found that salt solutions prevented Heat Exhaustion in men on the march and workers in hot industries, and the beneficial effect of salt in experimental Heatstroke has already been described (pp. 69-71). MacGregor(456) advises the feeding of sodium chloride for the prevention and treatment of Heatstroke and allied conditions in horses (see Appendix I). The more liberal use of salt might forestall the need for the excessive use of condiments and spices such as curry, to which much of tropical dwellers' indigestion is traditionally ascribed, since these are probably taken in response to achlorhydria, which may result from salt depletion. Condiments when taken in excess are irritating to the intestinal tract and even to the renal parenchyma, traces of condiments having been found in the urine(457). The importance attached to salt by natives of tropical countries is well known, and Julian Huxley(458) found that African children spend their "pocket money" not on sweets but on salt.

Gandhi/

Gandhi showed his wisdom when he made resistance to the Salt Tax one of the main offensives in his campaign against the British rule in India.

With a view to preventing the occurrence of Prickly Heat, which seems to be commonly associated with seborrhoeic conditions of the skin, I gave to a number of soldiers during the hot weather of 1932, an alkaline mixture containing sodium bicarbonate which Doble(459) recommended on account of his opinion which is held by many other authorities that seborrhoeic subjects suffer from a relative acidosis and are benefited by alkalies.(460) to (465) Those who took the alkaline mixture, many of whom had never avoided Prickly Heat during the hot weather, mostly remained quite free from the condition, and seemed also to be generally benefited in their ability to withstand heat. I have subsequently thought it possible that much of the benefit which they derived from it may have been due to the conversion of the sodium bicarbonate in the body to sodium chloride, as suggested by Marsh(82) in connection with alkalies used in the treatment of Heatstroke. Campbell(466) found salt very useful in the treatment of dermatitis due to irritation and profuse sweating, so the effect on the skin as well on the general condition may well have been due to sodium chloride.

Good cooking of the food is essential so that it/

it may be attractive and easily digestible, and although there are exceptions, army cooks are on the whole quite good and their efficiency is maintained by courses of instruction.

The importance of a sufficient supply of drinking water in hot climates, and its value in helping to prevent ill effects of heat have already been mentioned (pp.127-128) and medical authorities unite in stressing this point (2)(89)(150)(159)(160)(177)(222)(251)(309)(397)(467). When there is continued loss of fluid by sweating without adequate replacement a point must come at which, in order that the excretory function of the kidneys shall not fail, dehydration of the tissues must occur; (p.51-52:54) and as the kidneys are responsible for the excretion of some 35 grammes of waste material daily, if insufficient water is available for this purpose some retention of these products must occur.[468]

Huntington[325] says that in such places as the Death Valley in Arizona it is almost impossible to drink enough water to preserve normal physiological conditions, and Fremantle's[223] practice was to encourage men to count the half pints drunk in the day, and to aim at not less than a gallon, or in the hottest weather three gallons a day.

Although the medical offices may find that many commanding officers do not appreciate the need for/

for such large amounts of water there are usually no obstacles to the soldier obtaining enough drinking water in barracks.

It is however quite otherwise when he goes on a march, and his need for water becomes still more urgent. It has been found that on a march of 15 miles about 1000 Calories of heat require removal and since one Calorie is dissipated by the evaporation of 2 c.c. of water it follows that 2000 c.c. or four pints, which is roughly the contents of two water bottles, will be evaporated and should be replaced.[469] The medical officer will find that most commanding officers are unwilling to accept the simple rule that water lost in sweating must be replaced, their attitude to the subject being as archaic as that of the authorities in Philadelphia in 1814 who, under the mistaken impression that increased drinking of water was the cause rather than a symptom of Heatstroke, posted on the pumps warnings against sudden death from drinking water.[162] Martin,[21] who urged the drinking of a minimum quantity of three gallons a day, wrote "I found great difficulty in impressing this simple arithmetical calculation upon military authorities during some of our desert campaigning in 1916."

In Bareilly, when lecturing to officers, I stressed the importance of a generous supply of water/

water, and was treated by some senior officers as a dangerous heretic. A few weeks later when some of these officers came to a special pigsticking meet, a form of exercise to which they were unaccustomed, and one which produces a particularly intense thirst, I had my revenge when the water was brought up, by reminding them of their views, and begging them to set an example of which their men might be proud. I regret to have to say that they swallowed not only their principles but also a prodigious amount of water.

The official attitude according to the latest edition of Field Service Regulations[470] is that "the less men drink during the actual march the better, men should not be allowed to drink from their waterbottles without permission." Other sweating animals receive better treatment for, somewhat irrationally, the concluding sentence of the same paragraph reads "Every opportunity will be taken to water animals on the march."

An officer who has served with the Nigeria Regiment told me that on his first desert march with his company he ordered the usual restrictions on drinking water, and to his surprise the men, reputed to be wonderful marchers, stood up to the march very badly, most of them being exhausted, and many finishing/

finishing the march on transport vehicles. A senior native non-commissioned officer told him that if he allowed them to follow their usual practice of drinking freely there would be no further trouble. He took his advice, and the difference in the men's endurance was so marked that he followed their example and felt immeasurably the better for it. Anyone who has trekked with Himalayan hillmen, who are most particular as to the quality of the water they drink, will have noticed that they judge the severity of a day's march partly by the possibilities of obtaining frequent supplies of good water.

It would of course be unwise to assume that the military attitude to this subject is not based on experience, or to condemn it as unsound without further examination. I have been unable to elicit any reasons in its support except that it is "good for discipline", or that officers have known cases of men who drank freely and readily became exhausted or suffered from "colic." As regards the question of discipline, whilst it is true that soldiers may sometimes find themselves in circumstances in which strict conservation of water is essential, I cannot agree that it is wise to train them for such an eventuality by an unphysiological routine which exposes them to an added risk of Heatstroke.

Talbott[85] found that men accustomed to working in hot environments needed little extra fluid intake during/

during the hot months, although in unacclimatised people the fluid intake was greatly increased, and closely related to the elevation of the temperature. The trained soldier does not want to drink on the march nearly so much as the newcomer to India, but this should not be made a reason for limiting the water drunk by the latter, since acclimatisation is a gradual process which cannot and should not be hurried. The production of Heat cramps by drinking a large amount of water after profuse sweating has already been described, and also the fact that drinking water, even though it considerably relieves the symptons due to heat, may lead to excessive sweating (pp.65-70.). Both of these undesirable effects can however as we have seen be avoided by adding salt to the water which is drunk. The excessive sweating after the intake of water, which Lee and Mulder[63] considered to be partly reflex and partly absorptive in origin, caused Brodkin[104] to limit the intake of water by men on the march in order to prevent undue loss of sodium chloride, a factor which is itself an added cause of excessive sweating. By adding salt to the water which is drunk we can eliminate all the ill effects of water intake and avoid the necessity of impairing bodily hydration by withholding water, and in addition by replacing the salt already lost in sweating we are protecting the body from the effects/

effects of heat. There can be no doubt that this is the true solution of the problem and it only remains to be decided how it can be put into effect.

It has been found that 0.1 to 0.5 solutions of salt are not unpalatable and do not cause thirst,[62] whilst they do not, as stronger solutions may, lead to water retention or to raise the serum chloride too high (pp.163,164).

It should be possible by experiment to find the strength of saline solution which would be effective without proving distasteful to the soldier, and to provide tablets for addition to the water bottle, possibly flavoured with fruit juices. The use of these tablets should be necessary only on long marches in hot weather, as the moderate drinking of water which trained soldiers would find helpful under ordinary conditions should not be at all harmful.

A plentiful supply of ice is ensured by regulations and the medical officer can have the supply increased if necessary. Some supervision may be needed to ensure that units make the best use of the ration of ice, which is sufficient,if used wisely, to ensure that enough cold water is available for drinking at all times of the day or night as well as for use in ice boxes to preserve food. I am opposed to the practice of giving a proportion of the ice ration to the Regimental Contractor in order that he may supply/

supply iced drinks to the troops at a lower cost. Defaulters are likely to have the least money to spend on iced drinks, and as they usually do the most work their need of cold fluid is the greatest. One day after admitting three cases of Heatstroke who all said that the water in their barrack rooms during the night had been tepid or hot, I visited the lines and found blocks of ice which had weighed several pounds melting in the sun on the ration stand, because the contractor to whom they had been allotted had not called for them. It needed little imagination to realise that within the next twenty four hours men's lives might have depended on that puddle of warm water which should have been ice. The use of earthenware chatties for water instead of the galvanised iron tanks provided in some barracks helps to ensure a supply of really cold water for drinking during the night, but some of the ice ration should be set aside for this purpose.

Whilst stressing the importance of replacing fluid one should discourage those who replace it mainly with alcohol. (p.129). In a discussion of the subject by the United Services Medical Association in 1909[471] it was maintained that daily alcohol was not a necessity, but that a little might be taken as a drug when the body requires it, and that weak whiskey was the best, and malt liquors the worst/

worst form to take, an opinion endorsed by Morton[160]. Many people habitually take alcohol and seem to suffer no ill effects particularly if, in accordance with a wise tradition, it is not taken before the sun goes down and then only in moderation. Very rarely nowadays does one encounter a soldier who takes too much alcohol. Their usual choice is mineral water or tea, probably because these are cheap, since few will refuse the offer of an alcoholic drink. Although Fantus[397] advises the avoidance of coffee and tea there seems to be no adequate reason for prohibiting the latter, which as Sir Havelock Charles[471] said is an excellent drink "from the snows of the Pamirs to the scorching sands of Rajputana", and which others have found to be very suitable for military service.[389][472]

Drinker's[251] prescription for life in hot climates is a moderate amount of simple food, plenty of water, an adequate salt intake, no alcohol, adequate sleep and daily exercise. All these points have been discussed except the last two which will now be considered.

SLEEP and EXERCISE.

Adequate sleep is not always easily obtained during very hot weather, but much can be done by cooling the barrack rooms or by encouraging men to sleep in the open air at some distance from the buildings. In sleeping out of doors the officer has/

has an advantage as he can always hire a table fan to play upon his bed all night and some such arrangement might well be provided for the soldier.

It is unfortunate that, in order to get most of the work done before the heat of the day, men have to be woken up early just when, in the cool of the morning, they have reached the most refreshing hours of sleep offered by a hot-weather night. I consider this to be undesirable, and although it is certainly wise to reduce active physical work in the heat of the day, it should not be necessary to confine healthy men indoors from about 9 a.m. till 5 p.m. as is sometimes advised.[159] I frequently observed that whilst Infantry who were so confined were far from healthy, Gunners who worked in stables stripped to the waist during the morning, or exercised horses, seemed to thrive. In the Report on the Health of the Army for 1931 it was stated that the instructions regarding the "locking up" of troops in barracks during the greater part of the day were modified so as to permit of men being given something to do and that the general opinion was in favour of the new method.

In the Sanitary Report of the Army Medical Department for 1859 the value of exercise was emphasised and it was stated that "the want of exercise and monotony of a soldier's life in times of peace have/

have long been familiar to the medical officer as causes of disease. It is in India, however, that the baleful effects of indolent living are most conspicuously observed, indeed, caeteris paribus, it would not be difficult to prove that the measure of health which an Englishman enjoys is in direct relation to the amount of active exertion which he has to undergo." The soldier "has no object of sufficient interest to compel him to take much exercise, and few resources or amusements to tempt him out of doors: he yields to the seductions of an enervating exhausting climate, acquires sedentary, and too often dissipated habits, and deteriorates in physical and intellectual endowments." To counteract this indolence it was recorded with satisfaction that "Skittle grounds have been constructed for the use of the men, and quoits and draft boards supplied to each company at the expense of the Canteen fund." Although as Heald[473] points out there may be little scientific evidence to support the view that exercise prevents disease, there is general agreement that those who take regular exercise during the hot weather remain in better condition. This is probably because by open air exercise, good cardiovascular tone and active general metabolism are maintained and the skin is kept in a freely acting condition. Physical training in the early morning should/

~~Physical training in the early morning~~ should be a routine, and such strenuous exercise as football and hockey should be undertaken only in the cool of the evening. I have found it a good plan to recommend that games such as football should be divided, not into two even halves of half to three quarters of an hour, but into several "chukkers" of about ten minutes. The daily siesta from about 1.30 to 4.30 p.m. is an invaluable custom as it is often during that period that most of the daily sleep can be obtained, especially on hot dry days, probably as Hill[176] suggests because owing to the sun's heat convection currents are more active, and therefore in spite of the high dry-bulb temperature (The wet-bulb not being raised) conditions are more supportable. There is more movement of the air on the verandah, and the air which comes into contact with the body is relatively dry. Manoeuvres and long marches are not usually in peace time carried out during the hot weather, but even during the colder season conditions may be such as would lead to Heatstroke especially if there are added those factors which Pembrey[2] cited as making soldiers especially liable to Heatstroke. "The conditions", he says, "under which soldiers work are the most favourable for the production of Heatstroke; they are unsuitably clothed, heavily laden, and march in such close order that/

being carried on with a view to producing equipment which can be carried in the most physiological way, and by the separation of essentials from non essentials to ensure that as often as possible the soldier may be relieved of a large part of his load by mechanical transport. Although training improves the soldiers' load-carrying capacity, the trained man responding to the strain by a slight rise of blood pressure instead of, as does the untrained man, by a rise of pulse rate,[311] it does not increase his resistance to hot, moist, stagnant air,[309] and steps must be taken to exclude the other factors alluded to by Pembrey. Field Service Regulations and the Army Manual of Hygiene and Sanitation make wise recommendations for eliminating conditions likely to lead to Heatstroke on marches. Before a long march men are trained in marching, march discipline, and the adjustment of equipment in the most rational way, and their feet are carefully inspected to eliminate such conditions as blisters, the discomfort of which lead to unnecessary heat production. Night marches and very early starts are avoided if possible as they involve greater expenditure of energy and loss of sleep. Reveille is so timed that there shall be no unnecessary waiting about, and a light breakfast with hot sweet tea to provide a readily available carbohydrate source of energy is given. The pace should/

should be slow at the start to allow of adjustment, and thereafter a uniform pace is adopted, forced marches if necessary being achieved by increasing the duration of the march rather than the rate of marching. When serving with a Light Infantry regiment I frequently, though unsuccessfully, endeavoured to persuade them to abandon their customary speed of 115 to 120 paces to the minute when on the march. This speed, in which they take great pride, and even more so the "jog trot," of 140 to the minute at which they march past, is very trying to all but short men. Quite apart from the obvious unsoundness of jolting a possibly blistered foot some 5 to 10 times unnecessarily each minute, it has been proved that the normal load can be most economically carried with a long slow stride, although a very heavy load is more easily carried with a short stride.[311]

Monotony is eliminated by singing and the presence of the band. At the halts of ten minutes every hour men are encouraged to remove their equipment and to sit or lie down. On a forced march of 77 miles in 44½ hours Sykes[475] found it very helpful to get men to massage one another's legs, using firm kneading and passive movements, and I have found that many Himalayan hillmen use this method of increasing one's marching capabilities with excellent results in eliminating fatigue, and of course when one is thoroughly fatigued,/

fatigued, one walks clumsily, stumbling on stones and irregular ground and so increasing heat production.

To eliminate the humidity surrounding the column, to which Pembrey alluded, the column should be well spread out, two files should march on each side of the road, and at each halt the positions of companies in the column and of platoons in the companies should be changed, those marching in the rear coming to the front, so that the most adverse conditions, such as dust, shall not be confined to a few men who might thereby become quickly fatigued. Men should be allowed to drink from their water bottles at least after the first 7 or 8 miles, and saline solution may be used as already described. The medical officer assisted by the water duty personnel is responsible for the purity of the sources of water supply, and drinking from unauthorised sources is forbidden. Smoking should be reduced as much as possible, since it dries the mouth, and increases thirst, and it is usually allowed only at halts. On a long march a long halt of an hour is generally made about half way to the destination, and a midday meal may then be provided. On the completion of the march it is important in the Tropics that every man should be able to have a bath or a sponge down, and on arrival in camp hot sweet tea should be issued before the necessary fatigues are carried out, after which the main meal of the day is served/

During the march the medical officer should keep an eye on the men in order to detect any incipient cases of Heat Exhaustion or Heatstroke, and to forestall those referred to by Pembrey whose pride compels them to march "till they drop." Such a case occurred in a short march in summer in England when a man contrived to pass the barrack gates, supported on each side by his friends, only to drop unconscious and to die almost immediately. He was the man referred to before, (p. 178) whose death was probably due to Heatstroke, the only postmortem finding being slight cardiac dilatation. A medical officer who has taken pains to know the men of the regiment with which he is serving, who takes a deep interest in all that concerns their welfare, and has been rewarded by their trust and esteem, will be able by watchfulness on the march to prevent most of such unfortunate occurrences.

There are then many things which can be done to lessen the incidence of the Effects of Meat, and,to ensure their complete success, the unceasing vigilance of the medical officer is necessary. At all times there is nothing in the environment of the soldier which is too trivial to be worthy of the attention of the regimental medical officer, and particularly is this the case during "Heatstroke weather." He should see that a knowledge of the essential/

essential principles of the prevention of Heatstroke is possessed by all ranks and by their families, and that in his own domain, the hospital, all possible facilities for treatment are readily available. Although I have complained that on the subject of drinking on the march some commanding officers do not see eye to eye with medical theory, they are in most matters more than willing to hear and to act upon the advice of the medical officer, who is their responsible adviser on all that concerns the preservation of the health of the troops, and he may be sure that any practicable proposals which he may make in connection with the use of barracks, adjustments in the diet, or any other factor in the soldiers' environment will receive a sympathetic hearing and will usually be promptly put into effect.

Climatic conditions should be carefully observed and when they are such as to lead to Heatstroke, and especially during heatwaves, which are generally qhite short periods of only a few days' duration, precautions should be redoubled. All unnecessary work and fatigues in the heat of the day should be curtailed, plenty of water should be drunk, and the ice ration may be increased. Men may be warned to report at once any dryness of the skin, frequency of micturition, headache, giddiness or drowsiness. One need not, I think, anticipate any/

any unnecessary work after issuing such a warning, for the British soldier does not "panic" easily, whilst, in my experience,malingering, especially when the medical officer and the senior warrant and non-commissioned officers know the men well and are men of sound judgment, is extremely rare.

It will be as well to ensure that officers realise that during hot weather unexpected cases of insubordination or unusual behaviour may be cases for the Hospital rather than for the Guardroom. A careful watch should be kept upon all patients in hospital, and even perhaps, as Hearne[148] suggests,upon the men sleeping in barracks, whom he recommends should be inspected during the night, those who are sweating being safely disregarded. A useful warning sign, as Hill[177] observes, is the reaction of men to the fans, and if they feel more comfortable with these switched off, danger may be anticipated. The Katathermometer showed that if sweating is suppressed the use of the punkah results in a gain in heat so that whilst they are a valuable means of prophylaxis when men are sweating, they are a dangerous means of hastening Heatstroke when they are not. The patients' skins should be felt from time to time to detect suppression of sweating, and their temperatures may be taken two hourly or even hourly in the heat of the day. Morton[160] advises the use by all patients of lemonade containing 10 to 15 grains of potassium citrate to a glass, of which a pint should be given thrice daily.

It is certainly a source of satisfaction that the increasing interest taken in these conditions has in the past few years resulted in a considerable reduction in their incidence, and it is not too much to hope that by the energetic application of the measures for their prevention and treatment, which have been described still further reductions in incidence and mortality may be possible. Army medical officers have always shown great interest in this subject and they are in a position, by employing to the best of their ability, both the measures which have stood the test of time, and those more recently introduced, to render valuable service to the State by effecting a substantial reduction in the inefficiency and weakening of our forces resulting from heat; a weakening which, in the event of war, the British Army, so small in comparison with the vast armies of other great powers, could ill afford.

SUMMARY OF THE CONCLUSIONS ARRIVED AT IN THE THESIS.

Effects of Heat, even under modern conditions and in peace time, constitute a considerable menace to the health of the soldier in hot climates, and in this Thesis measures are described by which the incidence of these conditions may be lessened and their effects mitigated.

The Regulation of body temperature is discussed, and it is shown that in response to the need for heat loss, considerable amounts of water and of sodium chloride may be lost since the most effective means of cooling the body in high environmental temperatures, is the evaporation of sweat from its surface. The results of failure of the heat regulation are described under four headings:- Circulatory Insufficiency, Dehydration, Salt Loss and Hyperpyrexia.

The most important factors leading to such failure are atmospheric conditions unfavourable to the evaporation of sweat, especially a high relative humidity in association with a high environmental temperature, or if the humidity is low the presence of a scorching wind. Whilst Heatstroke can occur without any exposure to the sun, it is thought to be impossible in the light of our present knowledge to dismiss the possibility that sunlight may play a part in/

in the causation of these conditions.

The influence of subsidiary factors, such as unsatisfactory environmental conditions, failure to take the precautions, and to make the adjustments in the manner of living called for by residence in hot climates, and illhealth, is discussed and the general effects of hot climates on white men are briefly described.

Clinical conditions comprised under the heading of Effects of Heat are described in terms of the four main forms of failure in the adaptation of the body to heat which are mentioned above.

The primary indications for Treatment differ somewhat in these four conditions, but in all of them the water lost in sweating must be replaced. The same generalisation cannot be made in the case of the replacement of salt, since although such replacement is specifically indicated in cases of salt loss, and greatly benefits many cases of the other three types, there are certain contraindications to its administration in severe dehydration and in Heat Hyperpyrexia (see pp.156,157: 195,196).

Whatever its place in Treatment the provision of a generous supply of salt is invaluable in the prevention of all these conditions, and for the maintenance of health in hot climates since it is probable/

probable that minor degrees of hypochloraemia resulting from continued loss of sodium chloride in sweat without adequate replacement, is responsible for many of the adverse effects of hot climates.

The replacement of the large quantities of water lost in sweating is also of great importance, since without such replacement the body cannot maintain the degree of sweating required for cooling purposes, and the excretory functions of the kidney, except at the expense of the bodily hydration.

The practice of limiting the intake of water by soldiers on the march is therefore condemned and it is pointed out that any adverse effects from drinking water can be prevented by the addition of salt.

Other prophylactic measures include the education of the soldier in adapting himself to hot climates, the provision of suitable housing and clothing, and the regulation of his meals, his sleep, his work, and his play, in such a way that no avoidable strain is imposed upon his temperature-regulating mechanism.

APPENDIX I.

HEATSTROKE AND ALLIED CONDITIONS IN HORSES.

In a recent privately printed monograph "Non-Sweating, Heatstroke, and Kumri, and their inter-relationship in Horses in India" MacGregor[456] maintains that these three conditions in horses, and possibly also a fourth, Lichen tropicus or Prickly Heat, are in reality varying expressions in an ascending scale of the same affection.

NON-SWEATING DISEASE OF HORSES: which MacGregor suggests should be renamed Chronic Benign Hyperthermia, is geographically widespread in India and is most common in imported horses, though country bred horses also suffer from it. The deputy director of the Imperial Bureau of Animal Health informs me that it is probably the most important disease of race horses in the East, being, he has been told, very important in Singapore, where however the experience is that a period in the hills soon restores the affected horses to normal.

The coat loses its sheen, and often shows skin affections and loss of hair. Sweating is almost entirely absent, and there are accelerated respirations, a soft full pulse, sometimes intermittent, and often with a palpable thrill, and a rise of temperature. The mucous membrane of the mouth is soft, spongy and pale, with occasionally an icteric tinge; there is polyuria,/

polyuria, capricious appetite, and loss of condition. Drowsiness and staggering sometimes occur.

HEATSTROKE: (or Acute Hyperthermia) is so common in non sweating horses that the latter condition may be regarded as a Prodromal stage of Heatstroke. The clinical condition of Heatstroke in horses is very similar to that in human subjects, but the mortality is lower, 10% being the highest on record. But the horse is nevertheless less fortunate than the man, for a large number have to be destroyed after an attack of Heatstroke as complete recovery is rare. MacGregor finds that 100% show more or less distinct inco-ordination or weakness of the hind quarters or Kumri.

In KUMRI, or as MacGregor would prefer to call it Thermic Paraplegia, the horse has weakness of the hindquarters and difficulty in backing or in turning freely in a small circle. He may drag the hind and even the fore feet, and may sit down on his quarters if the backing test is persisted in. When trotting these horses swing their hind legs outwards without much flexion of the hocks.

Cases have been described of these three conditions existing at the same time in one animal, and in all cases there is a tendency for them to develop one after the other, so that Heatstroke can be regarded as a Hyperacute state of Non-sweating, and is liable/

liable to leave behind it effects on the nervous system such as paraplegia or Kumri. Not infrequently the final manifestation of the syndrome, Kumri, develops in a non sweater without the intervention of the Heatstroke stage.

Amongst the etiological factors are Heat and humidity, Salt loss and Thyro adrenal insufficiency, factors which play a part also in the causation of human Heatstroke and allied conditions.

In the report of the Veterinary Department of Burma for 1934 experiments are described which were directed to determining if an acid urine was an etiological factor. No evidence of this was obtained, but the problem is receiving further attention.

In Prevention sodium chloride feeding, and iron medication, both in massive doses, raising of the Vitamin C. content of the diet by the addition of germinated gram, improvement of general hygiene and grooming, and the use of electric fans for cooling, give good results.

APPENDIX II.

ATMOSPHERIC CONDITIONS AND INSTRUMENTS USED IN MEASURING THEM.

Atmospheric conditions depend upon Temperature, Pressure, Humidity, and Air Movement.

ATMOSPHERIC TEMPERATURE:

Two scales in common, use, Fahrenheit (Freezing 32° Boiling 212°) and Centigrade (Freezing 0° Boiling 100°).

Conversion Formulae F to C $(F - 32) \times \frac{5}{9}$

C to F $\left(\frac{C \times 9}{5}\right) + 32$

THERMOMETERS:

Mercury, Spirit, Maximum, Minimum, Six's (Maximum, & Minimum combined) Wet and Dry Bulb, Whirling Hygrometer (wet and dry bulb thermometers mounted together on a frame.)

The shade temperature is read on a thermometer hung in a louvred box, standing on legs 5 feet from the ground, 20 feet from the nearest building, with the side of the box facing North open.

The whirling hygrometer, a more delicate instrument than the wet and dry bulb thermometer, is whirled at arm's length for some 20 seconds and is then quickly read.

ATMOSPHERIC PRESSURE:

The atmosphere consists of a mixture of air and water vapour and extends from sea level to a height varying/

varying from 6 kilometres at the Poles to 16 kilometres at the Equator.

BAROMETERS:

MERCURY Dry air presses with great weight on the surface of mercury in the bowl and so forces the column of mercury higher up the tube.

ANEROID. The greater the atmospheric pressure the more do the sides of the hermetically sealed box collapse, and so compress an internal spring, and a system of levers translates this pressure into atmospheric pressure which is recorded on the dial as inches or millimetres of mercury.

Meteorological charts record pressure as isobars; i.e. lines joining places having the same atmospheric pressure expressed in millibars.

ATMOSPHERIC HUMIDITY:

SATURATION. When the air can hold no more water vapour in suspension it is said to be saturated with water vapour. The amount of water vapour necessary to cause saturation depends on the air temperature, (the higher the temperature the more moisture can the air hold).

ABSOLUTE HUMIDITY: The amount of water vapour actually present in the air is estimated by absorption hygrometers and expressed as grains per cubic foot.

RELATIVE HUMIDITY: is the most useful index of atmospheric humidity, and is the amount of water vapour/

vapour present in the air expressed as a percentage of the amount which would be necessary to cause saturation at that particular temperature.

DEW POINT. The temperature at which the amount of water vapour in the air causes saturation.

VAPOUR PRESSURE. The weight at the earth's surface of the aqueous vapour present in the air. Calculated by formulae based on wet and dry bulb temperatures, atmospheric pressure, and the latent heat of evaporation.

Relative Humidity can be determined by using Hygrometric Tables issued by the Meteorological Office of the Air Ministry, based on the fact that the depression of the wet bulb is proportional to the amount of moisture in the air. From these tables can be read off directly the Relative Humidity, the Vapour Pressure and the Dew Point from any reading of the Wet and Dry bulb Thermometer. Relative Humidity can also be calculated from Saturation Vapour Pressure tables by formulae based upon vapour pressure at Dew Point and at Dry Bulb; and Dew Point also can be calculated in this way.

Dew Point is also estimated from meteorological tables, and directly by Daniell's, Regnault's and Dine's hygrometers.

AIR MOVEMENT.

The Anemometer measures the rate of air entry into/

into a room. It consists of a propellor and a series of cog wheels operating a dial. To calculate also the amount of air entering the room the instrument is placed in an air inlet 2/5 of the diameter away from the periphery, and timed for a minute, the reading on the dial showing how many feet of air have passed the instrument in this time which gives the air velocity. If the area of the inlet in square feet is then multiplied by the velocity in feet per second, this equals the number of cubic feet of air per second entering the room.

Air movement is also calculated from Katathermometer readings by means of a nomogram (see later).

As the Katathermometer gives an average reading of air velocity Vernon and Manley (Medical Research Council Report No.100 1925) devised a portable "hot wire" anemometer to record rapid oscillations when these are important, and they estimated the temperature of air currents with a thermopile.

MEASUREMENT OF COOLING POWER OF THE AIR:

For this the three factors Temperature,Humidity, and Air Movement must be taken into account. Cooling Power may be estimated by:

(a) The Katathermometer (b) by Formula.

The Katathermometer:

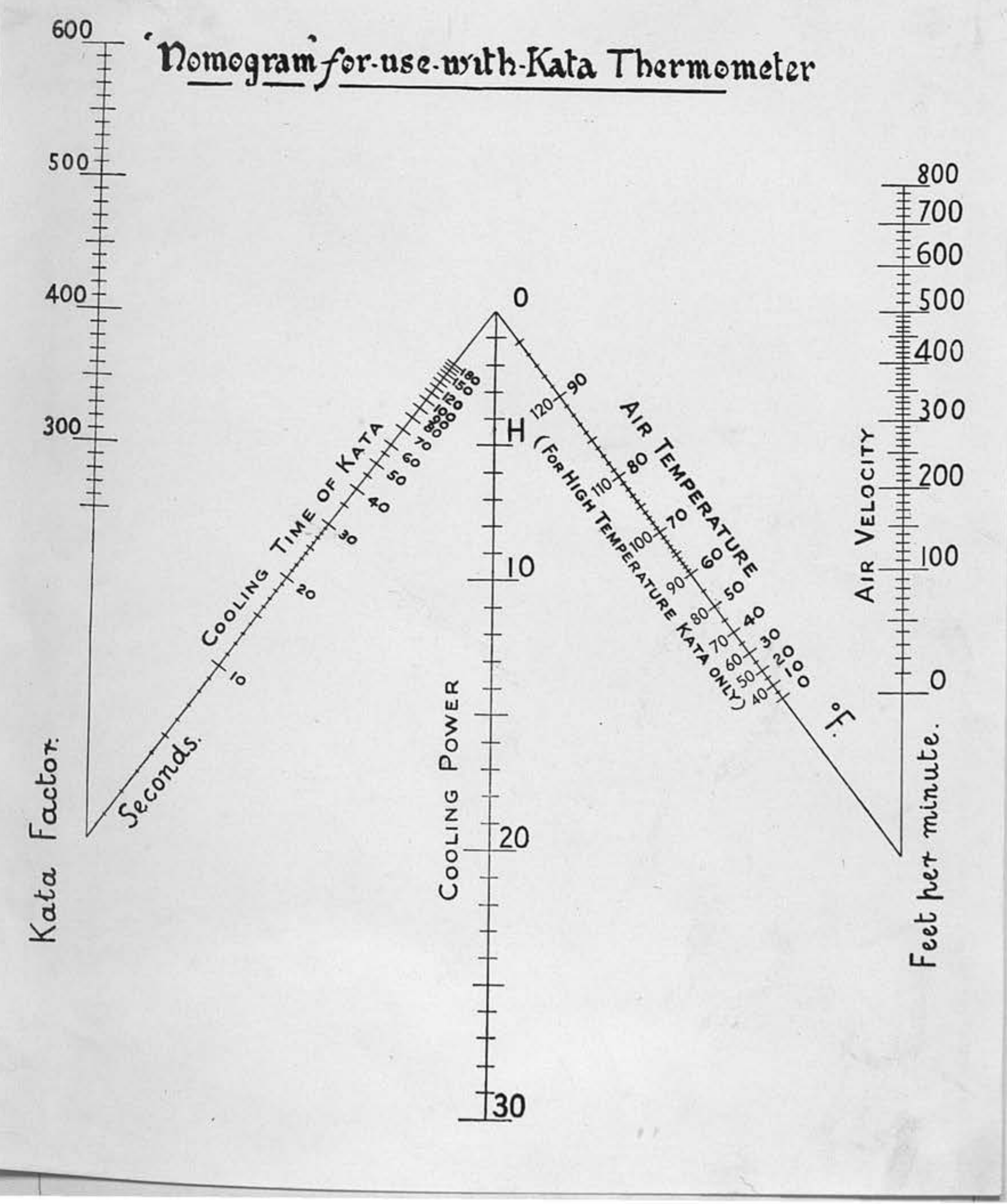
The Low Temperature Katathermometer is an alcohol thermometer with a large bulb and a stem graduated/

ted at 95° and 100°F.

The total heat lost per square centimetre of the surface of the bulb while cooling from 100° to 90°F. expressed in millicalories, is determined by the makers, and recorded on the stem, being known as the factor of the instrument. The instrument used with the bulb uncovered gives the Dry Kata reading, and covered with a wet, silk-net finger-stall it gives the Wet Kata reading.

The bulb is held in water heated to 150°F. and the alcohol rises into the small bulb. The large bulb is wiped dry and the instrument suspended well away from any source of heat or draught. The time taken for the alcohol to fall from 100°F to 90°F is noted with a stop watch. The first reading is discarded and a mean of five readings is taken. The factor divided by this mean gives the heat loss in millicalories per square centimetre of bulb surface per second - the Kata reading. The Kata reading may also be obtained by use of the nomogram shown below by making a line from the factor value on the factor scale to the cooling time in seconds on the time scale and producing this line to the cooling power scale, the point of intersection with which gives the Kata reading.

To estimate air velocity carry a line from the cooling power on the appropriate scale to the point on the Air Temperature scale corresponding to the air temperature at the time (being careful to use the scale designed for the low temperature kata) and thence to the air velocity scale on the extreme right where the velocity in feet per minute is read off. The High Temperature Katathermometer is used with the Nomogram to estimate air velocity when the temperature is above 90°F. A Recording Kata thermometer is described by Schuster in Report No.73 of the Medical Research Council (1923).



THE COOLOMETER:

A copper spool covered with a copper shell, the spool being wound with heating coils which constitute also an electrical thermometer. Used to study the cooling power, air movement, and radiation.

EFFECTIVE TEMPERATURE:

Chart showing influence of Dry bulb Temperature on Effective Temperature (Vernon & Warner)[220]

Dry Bulb T.	Wet Bulb T.	Effective T.	Rise in E.T. due to D.B.T.
70	70	70	
100	70	80.5	10.5
80	80	80	8.1
110	80	88.1	
90	90	90	5.5
120	90	95.5	

At higher temperatures the influence of the Dry Bulb Temperature is less important.

APPENDIX III.

THE SOLDIERS' LOAD.

NORMAL:

	Present lbs.	Present oz.	Experimental lbs.	Experimental oz.	
Clothing	14	9½	13	8	
Arms	10	7	10	7	
Ammunition	4	3	3	0	
Accoutrements	14	12	12	4½	- Reduction due to Steel helmet not being carried by man.
In the Pack	6	15½	5	0½	
Rations and Water	5	12	4	0	- Reduction due to change in emergency ration.

When in contact with the enemy:

Addition of more ammunition, + steel helmet, brings weights up to Normal 59 lbs. 11 oz. Experimental 51 7¾ (the lower pack being left with the Transport.)

The above weights do not include the great coat or entrenching tool, nor personal anti gas equip:ment with the exception of the respirator.

A very light experimental equipment weighing 34lbs. 12 ozs. is under trial. Opinion is in favour of a simple basic equipment comprising a belt and braces with one or more small ammunition pouches on each side, and a large haversack carried on the back containing waterbottle, mess tin and other essen:tials. Special pouches or other containers vary-

ing/

ing with the type of service would be attached to or slung over the equipment. The pack will normally be carried in unit transport.

APPROXIMATE WEIGHTS OF SOLDIERS' LOAD IN VARIOUS ARMIES:

	lbs.	oz.		lbs.	oz.
Great Britain	56	11	Norway	66	0
U.S.A.	62	8 to 74lbs.15 oz.	Sweden	64	0
Germany	82	0	Hungary	74	0
Italy	56	8	Bulgaria	61	12
Japan	58	0	Roumania	65	13
Russia	65	3	Czecho Slovakia	65	8
Turkey	64	8	Jugo Slavia	61	7

REFERENCES.

(Abbreviations those used in Index Medicus.)

1.	G. B. Malleson	History of the Indian Mutiny	1896.
2.	M. S. Pembrey & J. Ritchie	Text-book of General Pathology	1913.
3.	H. C. Bazett	Physiol Rev.	Oct. 1927
4.	S. Wright	Applied Physiology	1936
5.	J. Barcroft & A. V. Hill	J. Physiol	Mar. 1910
6.	F. A. Bainbridge	The Physiology of Muscular Exercise	1934
7.	C. H. Frazier, B. J. Alpers & F. H. Lewy	Brain	Mar. 1935
8.	S. W. Ranson & W. R. Ingram	Proc. Soc. Exp. Biol. & Med.	Jun. 1935
9.	L. M. Moore	Am. J. Physiol.	Jun. 1918
10.	H. G. Barbour	Brit. M. J.	Sep. 1913
11.	M. G. Eggleton	Muscular Exercise	1936
12.	J. B. Gaylor	Tr. Roy. Med. Chi. Soc. Glasgow	Nov. 1935
13.	W. Cramer	Fever, Heat Regulation, Climate & the Thyroid Adrenal Apparatus	1928
14.	C. R. Harington	The Thyroid Gland, its Chemistry & Physiology	1933
15.	D. Marine & E. J. Baumann	Am. J. Physiol	Feb. 1922
16.	J. Thomson	Clinical Study & Treatment of Sick Children	1921

17./

17.	H. Thursfield & D. Paterson	Diseases of Children	1934
18.	C. E. Lakin	Lancet	Sep. 1934
19.	G. Crile	Diseases peculiar to Civilised Man	1934
20.	D. H. K. Lee	Tr. Roy. Soc. Trop. Med. & Hyg.	June 1935
21.	C. J. Martin	Lancet	Sep. 1930
22.	C. D. Snyder	Am. J. Physiol	Mar. 1934
23.	N.Rakieten	Am. J. Physiol	Feb. 1936
24.	J. O. Balcar, W. D. Sansum & R.T.Woodyatt	Arch. Int. Med.	Nov. 1911
25.	A. Grollmann	Am. J. Physiol	Nov. 1930
26.	H. G. Barbour	Am. J. Physiol	Jan. 1924
27.	W. G. Maddock & F. A. Coller	Am. J. Physiol	Dec. 1933
28.	A. W. Hewlett, J. G. Van Zwaluwenburg, & M. Marshall	Arch. Int. Med.	Nov. 1911
29.	C. A. Johnson, S. Osborne & G. Scupham	Am. J. M.Sc.	Oct. 1935
30.	S. Nishibori a)	J. Orient Med.	Sep. 1934
31.	J. Adachi & S. Ito	J. Orient Med.	Dec. 1934
32.	Y. Kuno	The Physiology of Human Sweating	1934
33.	Y. Kuno	Lancet	Apr. 1930
34.	H. B. Richardson	J. Biol. Chem.	Feb. 1926
35.	R. W. Keeton	Am. J. Physiol	Jul. 1924
36.	J. J. R. Mac-Leod	Physiology in Modern Medicine	1935
37.	H. Iwatake	J. Orient Med.	Dec. 1934

38./

38.	K. Ogata	J. Orient Med.	Nov. 1934
39.	Gould's Medical Dictionary		1935
40.	H. H. Dale & W. Feldberg	J. Physiol	Aug. 1934
41.	C. L. Evans	Starling's, Principles of Human Physiology	1936
42.	K. Saito	J. Orient Med.	June 1935
43.	H. Cushing	proc. Nat. Acad. Sc.	Apr. May 1931
44.	E. H. Fishberg & W. Bierman	J. Biol.Chem.	Aug. 1932
45.	S. Nishibori b)	J. Orient Med.	Sep. 1934
46.	S. Ito & J. Adachi	J. Orient Med.	Nov. 1934
47.	K. Ogata	J. Orient Med.	June 1935
48.	L. Hill	Proc. Physiol Soc.	Feb. 1921
49.	T. Ichihashi & K. Ogata a)	J. Orient Med.	July 1935
50.	K. W. Kuo, K. Takahara, J. Adachi & K. Saito	J. Orient Med.	June 1935
51.	T. Ichihashi & K. Ogata b)	J. Orient Med.	July 1935
52.	W. H. Gordon & R. C. Jamieson	Ann. Int. Med.	Sep. 1931
53.	S. J. Thannhauser	J. A. M. A.	Mar. 1936
54.	J. R. Hodgkins	J. Roy. Army Vet. Corps	Nov. 1931
55.	L. Hill	Brit. M. J.	Mar. 1920
56.	B. A. McSwiney	Lancet	Mar. 1934
57.	E. H. Hunt	J. Hyg.	Dec. 1912

58.	O.R. MacEwen	Lancet	Apr. 1935
59.	E. F. Adolph	Am. J. Physiol	Nov. 1933
60.	S. Ito & T. Yabuki	J. Orient Med	June 1935
61.	K. N. Moss	Proc. Roy. Soc. London, S.B.	Aug. 1923
62.	C. P. McCord & T. L. Ferenbaugh	Mil. Surgeon	Dec. 1931
63.	D. H. K. Lee & A. G. Mulder	J. Physiol	Jul. 1935
64.	F. Marsh	Tr. Roy. Soc. Trop. Med. & Hyg.	Nov. 1930
65.	D. H. K. Lee & A. G. Mulder	J. Physiol	June 1935
66.	A.M.H. Grey	in Price's Text-book of Medicine	1937
67.	G. A. Talbert & C. O. Haugen	Am. J. Physiol	June 1927
68.	W. Hancock	reported in Brit. M. J.	Sep. 1929
69.	D. B. Dill, B. F. Jones, H. T. Edwards, & S. A. Oberg	J. Biol. Chem.	May 1933
70.	L. Hill & A. Campbell	Health & Environment	1925
71.	P. H. Manson-Bahr	Manson's Tropical Diseases	1935
72.	R. A. MacCance	Lancet	Mar. - Apr. 1936
73.	W. M. Marriott	Physiol Rev.	Apr. 1923
74.	R. C. Herrin	J. Biol. Chem.	Feb. 1935
75.	L. L. Mackay & E. M. Mackay	Am. J. Physiol	Oct. 1924
76.	A. G. Mitchell & L. Jonas	Am. J. M. Sc.	Feb. 1925

77./

77.	M. Maizels & J. Smith	Lancet	June 1934
78.	G. A. Talbert & I. Rosenberg.	Am. J. Physiol	Apr. 1928
79.	H. G. Barbour & W. Bourne	Am. J. Physiol	Jan. 1924
80.	D. W. Atchley	J. A. M. A.	Aug. 1930
81.	H. G. Barbour & E. Tolstoi	Am. J. Physiol	Jan. 1924
82.	F. Marsh	Tr. Roy. Soc. Trop. Med. & Hyg.	Nov. 1933
83.	R. Hutchison & D. Hunter	Clinical Methods	1929
84.	W. Hancock, A. G. R. White-house & J. S. Haldane.	Proc. Roy. Soc. Lond's B.	June 1929
85.	J. H. Talbott, H. T. Edwards, D. B. Dill,& L. Drastich	Am. J. Trop. Med.	Jul. 1933
86.	J. H. Talbott & J. Michelsen	J. Clin. Investi-gation	May 1933
87.	D. W. Atchley, R. F. Loeb, D. W. Richards, E. M. Benedict,& M. E. Driscoll	J. Clin. Investi-gation	Mar. 1933
88.	D. W. Atchley, & E. M. Benedict	J. Biol. Chem.	May 1927
89.	J. H. Talbott	Medicine	Sep. 1935
90.	S. A. Kofoed	Lancet	Jan. 1936
91.	M. S. Elliott	Mil. Surgeon	Mar. 1908
92.	R. J. W. Oswald	Lancet	June 1925
93.	R. Thrower	Brit. M. J.	Mar. 1928
94.	A. V. Elder	M. Press.	Oct. 1925

95.	D. M. Glover	J. Indust. Hyg. & Toxicol.	Dec. 1931
96.	D. B. Dill, A. V. Bock, H. T. Edwards, & P. H. Kennedy.	J. Indust. Hyg. & Toxicol.	Sep. 1936
97.	M. W. Heilman & E. S. Montgomery	J. Indust. Hyg. & Toxicol.	Nov. 1936
98.	W. B. Cannon	The Wisdom of the Body	1932
99.	F. R. Winton & L. E. Bayliss	Human Physiology	1936
100.	G. P. F. Allen	Brit. M. J.	June 1928
101.	F. Walinski	reviewed in Brit. M. J.	Sep. 1937
102.	H. G. Barbour, M. H. Dawson & I. Neuwirth	Am. J. Physiol	Sep. 1925
103.	S. Ito & T. Kosuge	J. Orient Med.	Jul. 1935
104.	H. A. Brodkin	Mil Surgeon	June 1934
105.	C. Van Zwalenburg	J. A. M. A.	Oct. 1933
106.	D. M. Kydd	J. Clin. Investigation	Nov. 1933
107.	C. F. Dixon	J. A. M. A.	May 1924
108.	W. D. Gatch, H. M. Trusler, K. D. Ayers	Am. J. M.Sc.	May 1927
109.	H. M. Trusler	J. A. M. A.	Aug. 1928
110.	T. G. Orr & R. L. Haden	J. A. M. A.	Nov. 1928
111.	M. A. McIver & J. L. Gamble	J. A. M. A.	Nov. 1928
112.	A. Miles & D. P. D. Wilkie	Thomson & Miles Manual of Surgery	1931

113.	H. Bailey & R. J. M. Love	A Short Practice of Surgery	1935.
114.	C. P. G. Wakely & J. B. Hunter	Rose & Carless Manual of Surgery	1937.
115.	W. Walters, A. M. Kilgore & J. L. Bollmann	J. A. M. A.	Jan. 1926
116.	H. Kretzschmer	reviewed in J. A. M. A.	Dec. 1934
117.	S. W. Smith	Brit. M. J.	Dec. 1937
118.	H. W. Barber & G. H. Oriel	Brit. M. J.	Nov. 1928
119.	J. H. Black & A. H. Braden	J. Allergy	Nov. 1936
120.	J. S. Haldane	Brit. M. J.	June 1923
121.	N. W. Mackeith M.S. Pembrey W. R. Spurrell E. C. Warner & H. J. W. J. Westlake.	Proc. Roy. Soc. London s B	Dec. 1923
122.	J. S. Haldane	Brit. M. J.	Apr. 1928
123.	J. Priestly	J. Physiol	Nov. 1921
124.	L. G. Rowntree	Am. J. Physiol	Feb. 1922
125.	C. H. Greene & L. G. Rowntree	Am. J. Physiol	Mar. 1924
126.	H. C. Gram	J. Biol. Chem.	Feb. 1924
127.	F. P. Underhill & M.A. Sellick	J. Biol. Chem.	Feb. 1924
128.	C. H. Greene & L. G. Rowntree	Am. J. Physiol	Mar. 1927
129.	F. S. Smyth, W. C. Deamer & N. K. Phatak	J. Clin. Investigation	Jan. 1933
130.	L. Rowntree	J. A. M. A.	Aug. 1923
131.	J. L. Miller & J.L. Williams	Am. J. M. Sc.	Mar. 1921

132./

132.	E. H. Derrick	Lancet (annotations)	Jan. 1935
133.	W.W. Swingle, W. M. Parkins, A. R. Taylor & H. W. Hays	Am. J. Physiol	July 1936
134.	W.W. Swingle, W.M. Parkins, & A.R. Taylor	Am. J. Physiol	July 1936
135.	F. G. Banting & S. Gairns	Am. J. Physiol	June 1926
136.	W. Langdon Brown	Lancet	May 1935
137.	W. Oakley	Lancet	Jul. 1935
138.	M. Maizels & C. B. MacArthur	Quart J. Med.	Jul. 1929
139.	J. G. Adami	Principles of Pathology	1908
140.	J. Coats	Manual of Pathology	1903
141.	D. J. Hamilton	Text-boox of Pathology	1894
142.	M. Flack & L. Hill	Text-book of Physiology	1919
143.	R. Tigerstedt	Text-book of Human Physiology	1906
144.	D. Noël Paton	Essentials of Human Physiology	1920
145.	E. A. Schäfer	Text-book of Physiology	1898
146.	W. G. MacCallum	A Text-bbok of Pathology	1936
147.	R. Burton Opitz	Text-book of Physiology	1920
148.	K. G. Hearne	Brit. M. J.	Apr. 1919
149.	K. G. Hearne	Brit. M. J.	Apr. 1920
150.	L. Rogers, & J. W. D. Megaw	Tropical Medicine	1935
151.	W. Pike	Brit. M. J.	Apr. 1920

152./

152.	A. H. H. Howard	Brit. M.J.	Apr. 1920
153.	R. J. M. Love	Brit. M. J.	June 1919
154.	E. B. Ferris	J. Medicine	Jan. 1937
155.	K. G. Hearne	Brit. M. J.	Aug. 1919
156.	G. Graham & E. P. Poulton	Quart. J. Med.	Oct. 1912
157.	H. G. Barbour	Physiol. Rev.	Apr. 1921
158.	H. Sutton	J. Path & Bact.	1930
159.	W. H. Willcox	Brit. M. J.	Mar. 1920
160.	T. C. St.C.Morton	J. Roy. Army M. Corps.	Sep. 1932
161.	H. Heller & F. H. Smirk	J. Physiol	Sep. 1932
162.	E. G. Wakefield, & W. W. Hall	J. A. M. A.	Jul. 1927
163.	W. M. Bayliss	Principles of General Physiology	1920
164.	E. R. Stitt	Diagnostics & Treatment of Tropical Diseases	1929
165.	A. G. Mayer	Am. J. Physiol	Nov. 1917
166.	D. Livingstone	Missionary Travels & Researches in S. Africa p. 652	1857
167.	J. J. Levick	Am. J. M. Sc.	Jan. 1859
168.	W. Byam & R. G. Archibald	Practice of Medicine in the Tropics	1923
169.	A. Castellani & A. J. Chalmers	Manual of Tropical Medicine	1919
170.	W. C. Brown	Brit. M. J.	June.1906
171.	J. F. Payne	Manual of General Pathology	1888
172.	C. Blagden	Phil. Tr. Roy. Soc. London.	Feb. 1774
173.	M. Dobson	Phil. Tr. Roy. Soc. London.	June 1775

174.	C. Blagden	Phil. Tr. Roy. Soc. London.	Jul. 1775
175.	H. Stott	Indian M. Gazette	Dec. 1936
176.	L. Hill	M. Res Committee report 32	Jan. 1919
177.	L. Hill	M. Res. Council report 52	1920
178.	G. Mouriquand & R. Charpentier	Montpellier Méd	Jul. 1936
179.	L. Hektoen & D. Riesman	Textbook of Pathology	1901
180.	L. Landois	Textbook of Human Physiology	1904
181.	L. Rogers	J. Roy Army M. Corps	Jan.1908
182.	G.N. Stewart	Manual of Physiology	1914
183.	L. Rogers	Fevers in the Tropics	1919
184.	L. Luciani	Human Physiology	1921
185.	R. J. S. Simpson	J. Roy Army M. Corps	May 1923
186.	W. F. Dearden	J. Indust Hyg. & Toxicol	Sep. 1927
187.	W. Boyd	Textbook of Pathology	1934
188.	H. T. Karsner	Human Pathology	1935
189.	H. E. Roaf	Textbook of Physiology	1936
190.	E. Lozinsky	Am. J. Physiol	Jan. 1924
191.	J. S. Haldane	J. Hyg	Oct. 1905
192.	F. R. Winton & L. E. Bayliss	Human Physiology	1936
193.	W. H. Carrier	reviewed in Bulletin of Hygiene	1930
194.	T. Bedford	J. Hyg	Aug. 1935
195.	W. Heberden	Phil. Tr. Roy. Soc. London.	Jan. 1826

196./

196.	L. Hill, O. W. Griffith & M. Flack	Tr. Roy. Soc. London s B.	June 1916
197.	L. Hill	J. Scientific Research	1930
198.	T. C. Angus, L. Hill & H. C. Soper	J. Indust. Hyg. & Toxicol.	Feb. 1930
199.	C. P. Yaglou K. Dokoff	J. Indust. Hyg. & Toxicol.	Oct. 1929
200.	W. S. Weeks	J. Indust Hyg. & Toxicol	Apr. 1930
201.	W. S. Weeks	J. Indust. Hyg. & Toxicol.	Sep. 1931
202.	C. Iwabuchi	Taiwan Igakkai Zasshi	Apr. 1934
203.	C. P. Yaglou	J. Indust. Hyg. & Toxicol.	Sep. 1935
204.	D. Hargood Ash & L. Hill	M. Res Council Report 73	1923
205.	H. M. Vernon	M. Res Council Report 100	Dec. 1925
206.	T. Bedford	J. Indust. Hyg. & Toxicol	Dec. 1928
207.	L. Hill, T.C. Angus & E.M. Newbold	J. Indust. Hyg. & Toxicol	Dec. 1928
208.	T. Bedford & C. G. Warner	J. Indust. Hyg. & Toxicol	Apr. 1931
209.	C. P. Yaglou	J. Indust. Hyg. & Toxicol	Jan. 1926
210.	C. P. Yaglou	J. Indust. Hyg. & Toxicol	Sep. 1926
211.	C. P. Yaglou	J. Indust. Hyg. & Toxicol	Jul. 1927
212.	C. P. Yaglou & P.Drinker	J. Indust. Hyg. & Toxicol	Dec. 1928
213.	R. C. Partridge & D.L.MacLean	J. Indust. Hyg. & Toxicol	Mar. 1935

214./

214.	C. P. Yaglou	J. Indust. Hyg. & Toxicol	June 1927.
215.	H. M. Vernon	J. Indust. Hyg. & Toxicol	Sep. 1926.
216.	H. M. Vernon	J. Indust. Hyg. & Toxicol	Jul. 1927.
217.	J. A. Campbell & T. C. Angus	J. Indust. Hyg. & Toxicol	Dec. 1928.
218.	T. Bedford & C. G. Warner	J. Indust. Hyg. & Toxicol	Apr. 1931.
219.	H. M. Vernon & T. Bedford	J. Indust. Hyg. & Toxicol	Feb. 1927.
220.	H. M. Vernon & C. G. Warner	J. Hyg.	Jul. 1932.
221.	H. M. Vernon	J. Indust. Hyg. & Toxicol	Nov. 1932.
222.	F. H. G. Hutchinson	Ind. J. M. Research	Jul. 1926.
223.	F. E. Fremantle	Lancet	Sep. 1919.
224.	M. S. Pembrey	J. Roy. Army M. Corps.	Aug. 1913.
225.	C. S. P. Hamilton, D.N.Basu, & J.Van Haeften,	J. Roy. Army M. Corps.	Aug. 1930.
226.	L. J. Llewellyn	M. Press.	Nov. 1930.
227.	A. Davidson	Hygiene & Diseases of Warm Climates	1893.
228.	H. S. Corbusier	Mil Surgeon	June 1908.
229.	T. K. Monro	Manual of Medicine	1925.
230.	C. Joyeux	Précis de Médicine Coloniale	1927.
231.	J. N. Dugdale	Health in Hot Climates	1931.
232.	J. Henderson	Wheeler & Jack's Handbook of Medicine	1932.
233.	H. L. Tidy	Synopsis of Medicine	1934.

234./

234.	R. Muir	Textbook of Pathology	1936.
235.	H. C. Wood	Fever	1880.
236.	L. W. Sambon	J. Trop. Med. & Hyg.	Feb. 1907.
237.	A. Duncan	J. Trop. Med. & Hyg.	Mar. 1907.
238.	A. Duncan	J. Roy. Army. M. Corps.	Jul. 1908.
239.	P. Manson-Bahr.	R. Hutchison's Index of Treatment.	1931.
240.	J. D. Phalen	J. Roy. Army M. Corps (reprint from Mil Surgeon)	Jan. 1911.
241.	R. J. Blackham	J. Roy. Army M. Corps.	Dec. 1913.
242.	J. R. Taylor	Lancet	Aug. 1858.
243.	R. J. S. Simpson	J. Roy.Army M. Corps.	Nov. 1908.
244.	R. J. S. Simpson	J. Roy. Army M. Corps.	Mar. 1909.
245.	W. H. Ogilvie	J. Roy. Army M. Corps	Oct. 1912.
246.	P. Mackenzie & E. R. Le Count	J. A. M. A.	Jul. 1918.
247.	L. Hill & J. A. Campbell	Lancet	Apr. 1923.
248.	E. S. Sundstroem	Physiol Rev.	Apr. 1927.
249.	H. Laurens	Physiol Rev.	Jan. 1928.
250.	J.J. Conybeare	Text-book of Medicine	1935.
251.	C. K. Drinker	J. Indust. Hyg. & Toxicol.	Oct. 1936.
252.	W. H. Willcox	in 'Price's Text-book of Medicine	1937.
253.	F. G. Bushnell	J.Roy.San.Inst.	Nov. 1928.

254.	L. Hill	M. Press	June 1930.
255.	L. Hill	Brit. J. Phys. Med.	Sep. 1931.
256.	J.J.R. MacLeod A.R. Self, & N.B. Taylor	Lancet	Sep. 1920.
257.	J.J.R.MacLeod & N.B. Taylor	Lancet	Jul. 1921.
258.	A. Eidinow	Brit. M. J.	Aug. 1929.
259.	A. Eidinow & L. Hill	Brit. M. J.	Mar. 1930.
260.	A. Eidinow	J. Path & Bact.	1930.
261.	A. Eidinow	J. Roy. San. Inst.	Nov. 1928.
262.	C.E. Woodruff	The Effects of Tropical Light on White Men	1905.
263.	C. Eijkman	Lancet	May 1924.
264.	F.C. Koch & C.I. Reed	Am. J. Physiol.	Jan. 1926.
265.	R. Aitken	Brit. J. Phys. Med.	June 1935.
266.	R.A. Johnston	Brit. M.J.	Nov. 1937.
267.	W.P. Kennedy	Brit. M. J.	Dec. 1937.
268.	A. Webster L. Hill, & A. Eidinow	Lancet	Apr. 1924.
269.	H.M. Vernon	J.Indust. Hyg. & Toxicol	Mar. 1932.
270.	L. Hill & A. Eidinow	Proc. Roy. Soc. London s B.	Aug. 1923.
271.	A. Aimes,	Montpellier Med.	Jul. 1936.
272.	H. Jausion	Montpellier Med.	Jul. 1936.
273.	L. Hill	M. Press	Jun. 1930.
274.	M. Roch	Presse Med.	Apr. 1936.
275.	S.W. Smith	Brit. M.J.	July 1934.
276.	S. Bray	M.J. Australia	Nov. 1933.

277.	L. Hill & J. A. Campbell	Brit. M. J.	Mar. 1922.
278.	K. R. C. Hallowes	Brit. J. Phys. Med.	June 1935.
279.	L. Mackey & A. E. Garrod	Quart. J. Med.	Jul. 1922.
280.	A. T. Cameron & C. R. Gilmour	Biochemistry of Medicine	1933.
281.	V. R. Mason, C. Courville, & S. Siskind	Medicine	Dec. 1933.
282.	L. Hill & J. A. Campbell	Brit. M. J.	Feb. 1922.
283.	R. KING BROWN	Brit. J. Phys. Med.	Mar. 1934.
284.	I. S. Falk & C. I. Reed	Am. J. Physiol	Feb. 1926.
285.	F. Sugden	Brit. J. Phys. Med.	Jan. 1935.
286.	P. Bauwens	Brit. J. Phys. Med.	May 1935.
287.	J. H. Clark	Am. J. Physiol	Nov. 1918.
288.	J. H. Clark	Physiol Rev.	Apr. 1922.
289.	C. I. Reed	Am. J. Physiol	Aug. 1923.
290.	C. I. Reed	Am. J. Physiol	Nov. 1925.
291.	W. A. Tatchell	Brit. M. J.	Sep. 1937.
292.	L. J. Llewellyn & A. Bassett Jones	Brit. J. Phys. Med.	Dec. 1931.
293.	V. Z. Cope	Brit. M. J. p. 401.	Mar. 1920.
294.	H. Gauss & K. A. Meyer	Am. J. M. Sc.	Oct. 1917.
295.	A. F. Hurst	Constipation and Allied Intestinal Disorders	1919.
296.	A. F. Hurst	in Price's Textbook of Medicine	1937.

297.	G. E. Beaumont	Medicine	1935.
298.	J. S. MacMillan	J. Roy. Army M. Corps	Feb. 1934.
299.	W. C. Alvarez	J. A. M. A.	Dec. 1917.
300.	W. C. Alvarez	J. A. M. A.	Jan. 1919.
301.	W. C. Alvarez	Physiol Rev.	Jul. 1924.
302.	W. Osler & T. McCrae	Principles & Practice of Medicine	1935.
303.	F. C. Wood	Text-book of Pathology (Delafield & Prudden)	1936.
304.	W. D. Halliburton	Handbook of Physiology	1920.
305.	T. Bedford	J. Hyg.	Aug. 1935.
306.	L. Hill	J. Roy. Army M. Corps.	Aug. 1913.
307.	W. H. Howell	Text-book of Physiology	1924.
308.	Reader's Digest		Dec. 1936.
309.	Report of Committee on Food &c.	J. Roy. Army M. Corps.	Feb. 1908.
310.	A. G. Stevenson &.R.L. Brown	J. Roy. Army M. Corps.	May - Aug. 1923.
311.	E. P. Cathcart, D. T. Richardson & W. Campbell	J. Roy. Army M. Corps.	July 1923.
312.	T. F. Kennedy	J. Roy. Army M. Corps.	July - Sep. 1933.
313.	C. E. H. Milner	Brit. M. J.	June 1918.
314.	C. H. L. Meyer	Brit. M. J.	Oct. 1918.
315.	H. Beadnell	Brit. M. J.	June 1918.
316.	H. C. Sinderson	Brit. M. J.	Jul. 1919.

317.	L. W. Sambon	Brit. M. J.	Mar. 1898.
318.	P. Manson	Tropical Diseases	1918.
319.	K. Macleod	Brit. M. J.	Sep. 1899.
320.	M. Fog	J. A. M. A.	Dec. 1936.
321.	G. C. Shattuck & M.M.Hilferty	New England J. Med.	Mar. 1936.
322.	K. Black	Malayan M. J.	Dec. 1932.
323.	A. Rattray	Proc. Roy. Soc. London	Feb. 1871.
324.	A. Rattray	Proc. Roy. Soc. London	Nov. 1872.
325.	E. Huntington	Civilization and Climate	1915.
326.	T. S. Rippon	Roy. Soc. Med. (War Section)	Apr. 1923.
327.	H. W. Acton	Ind. M. Gaz.	Jan 1927.
328.	G. W. Will	J. Roy. Army M. Corps.	Nov. 1937.
329.	M. Culpin	Lancet	Feb. 1933.
330.	D. K. Henderson & R. D. Gillespie	Text-book of Psychiatry	1930.
331.	D. V. Latham	East African M. J.	Jan. 1933.
332.	W. Lohman	Disturbances of the Visual Function	1913.
333.	W. R. Aykroyd	J. Hyg.	Aug. 1930.
334.	J. R. Mutch & H. D. Griffith	Brit. M. J.	Sep. 1937.
335.	F. P. Maynard	Manual of Ophthalmic Practice	1920.
336.	W. Wick	reviewed in J. Roy. Army M. Corps	Mar. 1911.
337.	D. M. Lyon & H. L. Wallace	Brit. M. J.	May 1932.
338.	J. H. P. Paton	Brit. M. J.	Jul. 1932.
339.	A. Breinl & H. Priestley	Ann. Trop. Med. & Parasitology	Dec. 1914.

K

339 omitted

340.	W. E. Cooke & E. Ponder	The Polynuclear Count	1927.
341.	W. E. Cooke	Clinical Interpretation of Aids to Diagnosis	1930.
342.	A. Piney	Recent Advances in Haematology	1931.
343.	W. P. Kennedy	Quart J. Exper. Physiol.	1933.
344.	A. Breinl & H. Priestley	Ann Trop Med. & Parasitology	Dec. 1915.
345.	A. Breinl H. Priestley	Ann Trop. Med. & Parasitology	Feb. 1917.
346.	W. P. Kennedy	Tr. Roy. Soc. Trop. Med. & Hyg.	Mar. 1935.
347.	W. P. Kennedy & I. Mackay	Tr. Roy. Soc. Trop. Med. & Hyg.	Nov. 1935.
348.	W. P. Kennedy & K. N. Flint	Quart J. Exper. Physiol	1930.
349.	W. P. Kennedy & K. N. Flint	Quart. J. Exper. Physiol.	1927.
350.	W. M. Shanklin	Tr. Roy. Soc. Trop. Med. & Hyg.	Jul. 1936.
351.	J. W. S. MacFie	Ann Trop. Med. & Parasitology	Dec. 1915.
352.	J. W. S. MacFie	Lancet	May 1915.
353.	H. C. Pai	J. Path & Bact.	Mar. 1935.
354.	J. MacLeod	Am. J. Physiol	Apr. 1935.
355.	R. W. Barris & W. R. Ingram	Am. J. Physiol	Feb. 1936.
356.	L. H. Roddis & G. W. Cooper	J. A. M. A.	Dec. 1926.
357.	H. A. Treadgold	Lancet	Apr. 1933.
358.	A. Rattray	Proc. Roy. Soc. London.	June 1870.
359.	W. Denis & P. Borgstrom	J. Biol. Chem.	Aug. 1924.

360.	C. Dobell	M. Res. Council Report 59	1921.
361.	I. MacDonald	Lancet	Dec. 1931.
362.	C.C. Lund & T. R. Ingham	J. A. M. A.	Nov. 1933.
363.	C. F. Craig	J. A. M. A.	Oct. 1934.
364.	M. S. Dougherty	Am. J. Trop. Med.	May 1933.
365.	T. Kawai, Y. Nagayoshi & C. Koo	Taiwan Igakkai Zasshi	Aug. 1934.
366.	E. W. Dennis & E. E. Lund	Tr. Roy. Soc. Trop. Med. & Hyg.	Jan. 1937.
367.	C. J. H. Little & W. Bornshin	J. Roy. Army M. Corps.	Dec. 1930.
368.	H. W. Acton	Tr. Roy. Soc. Trop. Med. & Hyg.	Jul. 1933.
369.	S. K. Simon	J. A. M. A.	Oct. 1934.
370.	G. T. Burke	Ind. M. Gaz.	Oct. 1933.
371.	J. Heatley Spencer	J. Roy. Army M. Corps.	Feb. 1934.
372.	K. M. Lynch	J. A. M. A.	Oct. 1934.
373.	W. Wilkinson	B. M. J.	Sep. 1935.
374.	J. Huston	J. Roy. Army M. Corps.	Sep. 1937.
375.	J. G. Thomson	Practitioner	July 1932.
376.	I. J. Kligler	Tr. Roy. Soc. Trop. Med. & Hyg.	Feb. 1936.
377.	A. I. Kendall	J. Med. Res.	Sep. 1911.
378.	Med. Res. Council	System of Bacteriology in relation to Medicine	1931.
379.	A. F. Hurst	Lancet	Jan. 1923.
380.	F. A. Knott	Guy's Hosp. Rep.	1923.

381./

381.	J. W. S. Blacklock K. J. Guthrie & I. Macpherson	J. Path & Bact.	Mar. 1937.
382.	A. F. Hurst	Brit. M. J.	Oct. 1937.
383.	F. E. Camps	Guy's Hosp. Rep.	1933.
384.	L. G. Brown	Brit. M. J.	Oct. 1937.
385.	J. Hare	J. Roy. Army M. Corps.	Nov. 1937.
386.	E. B. Waggett	J. Roy. Army M. Corps.	Nov. 1937.
387.	R. L. Benson	J. Allergy	Nov. 1936.
388.	J. H. T. King 1877	reprinted in Mil Surgeon	Apr. 1934.
389.	G. W. Steevens	With Kitchener to Khartoun	1898.
390.	A. F. Hartmann	Am. J. Dis. Child.	Apr. 1928.
391.	L. A. Hoag & E. Marples	Am. J. Dis. Child.	Aug. 1931.
392.	A. F. Hartmann & R. Elman	J. Exper. Med.	Sep. 1929.
393.	D. L. Edsall	Am J. M. Sc.	Dec. 1904.
394.	H. M. Welsh	J. A. M. A.	Apr. 1909.
395.	W. H. Cameron	J. A. M. A.	May 1909.
396.	D. L. Edsall	J. A. M. A.	Dec. 1908.
397.	B. Fantus	J. A. M. A.	Sept. 1934.
398.	F. G. Gardner	Brit. M. J.	Mar. 1928.
399.	S. D. Hine	Lancet	Nov. 1888.
400.	P. M. Starkov & J. V. Jikesh	J. Indust. Hyg. & Toxicol	Nov. 1935.
401.	A. Barclay	in San Rep. of Army Med. Dep.	1859.
402.	J. Bonnyman	Edinburgh M. J.	May 1864.
403.	H. A. Foy	Tr. Roy. Soc. Trop. Med. & Hyg.	Nov. 1930.

404.	C. A. Neymann & S. L. Osborn	J. A. M. A.	Jan. 1931.
405.	C. A. Neymann	Brit. J. Phys. Med.	Oct. 1931.
406.	H. Vesell & W. Bierman	Am. J. M.Sc	Apr. 1936.
407.	Memoranda on Med. Dis. in Tropical & Subtropical Areas (HM.Stationery Office)		1930.
408.	F. C. Hotz	Am. J. M. Sc.	Jul. 1879.
409.	R. M. Stewart	Rev. Neur. & Psych.	1918.
410.	Medical History	War of the Rebellion	1888.
411.	A. F. Messiter	Lancet	June 1897.
412.	T. H. Weissenburg	J. A. M. A.	June 1912.
413.	J. Finlayson	Clinical Manual	1891.
414.	W. P. MacArthur	Tr. Roy. Soc. Trop. Med. & Hyg.	Jan. 1934.
415.	E. B. Marsh	J. Roy. Army M. Corps	Apr. 1934.
416.	F. Holmes	J. Roy. Army M. Corps	Apr. 1934.
417.	H.B.F. Dixon & D. W. Smithers	"	June 1934.
418.	H.B.F. Dixon & D.W. Smithers	Quart. J. Med.	Oct. 1934.
419.	H.B.F. Dixon & D.W. Smithers	J. Roy. Army M. Corps.	Apr. to Aug. 1935.
420.	F. M. Lipscomb	J. Roy. Army M. Corps.	Dec. 1935.
421.	S.J.L.Lindeman & R.S.Lyburn	"	Aug. 1935.
422.	W. K. Morrison	"	Jan. 1936.
423.	R.B.H. Gradwohl & E. Schisler	Am. J. M. Sc	Sep. 1917.
424.	J. N. McIntosh	Brit. M.J.	June 1920.
425.	D. C. Rennie	Tr. Roy. Soc. Trop. Med. & Hyg.	Apr. 1930.

426.	R.A. Anderson & J.C. Sproule	J. Roy. Army M. Corps.	Oct. 1933.
427.	K. Dodd & S.J. Wilkinson	J. A. M. A.	Mar. 1927.
428.	F.A. Packard	Am. J. M. Sc.	June 1888.
429.	M.S. Pembrey	J. Roy. Army M. Corps	June 1913.
430.	B. Darrach	Am. J. M. Sc.	Jan. 1859.
431.	E.P. Poulton	Taylor's Practice of Medicine	1930.
432.	N. Woodyatt	Under Ten Viceroys	1922.
433.	L. Rogers	J. Roy. Army M. Corps.	June 1908.
434.	H.G. Barbour	Arch. Int. Med.	Dec. 1919.
435.	H.G. Barbour & M.M. Devern's	Arch Int. Med.	Dec. 1919.
436.	W.G. Maddock & F.A. Coller	J. A. M. A.	Jan. 1927.
437.	F.A. Coller, V.S. Dick & W.G. Maddock	J. A. M. A.	Nov. 1936.
438.	M. A. Falconer & A. Lyall	Brit. M. J.	Dec. 1937.
439.	W. Cramer	Brit. J. Exper. Path.	Feb. 1920.
440.	H. Willoughby & E. Aslett	J. Roy. Naval M. Serv.	Jan. 1932.
441.	J.J. Redmond	Lancet	Feb. 1932.
442.	P.B. Giles	Brit. M.J.	Sep. 1906.
443.	L.B. Rawling	Brit. M.J.	May 1918.
444.	G.P. Crowden	Lancet	Jan. 1934.
444a.	T.O. Thompson	J. Roy. Army M. Corps	May 1933.
445.	" "	J. Roy. Army M. Corps.	Feb. 1934.
446.	Report of Committee on Food Training etc. J. Roy. Army M. Corps.		Nov. 1909.

447./

447.	C. Iwabuchi	Taiwan Igakkai Zasshi	Apr. 1934.
448.	T. Onogi	Taiwan Igakkai Zasshi	Apr. 1934.
449.	A. Maurois	Times Supplement	Oct.26th 1937.
450.	B. Rush	Sep.1777 reprinted in Mil Surgeon	Mar. 1908.
451.	C. Watson	Food & Feeding in Health & Disease	1910.
452.	G. Lusk	The Elements of the Science of Nutrition	1917.
453.	P. Manson & C. W. Daniells	in System of Diet and Dietetics (Sutherland)	1908.
454.	W.R. Aykroyd & B. G. Krishman	Indian J.M.Research	Oct. 1936.
455.	W.R. Aykroyd	Brit. M.J.	Nov. 1937.
456.	A. D. MacGregor	Non Sweating Heat-stroke & Kumri	1936.
457.	M.A. Bridges & H.O. Mosenthal	Dietetics for the Clinician	1935.
458.	J. Huxley	Africa View	1931.
459.	F. C. Doble	Lancet	Feb. 1925.
460.	H.W. Barber & H.C. Semon	J. Roy. Army M. Corps.	Sep. 1919.
461.	H.W. Stelwagon & H.K. Gaskell	Treatise on Diseases of the Skin	1921.
462.	H.W. Barber	Lancet	Feb & Mar. 1925.
463.	C. Albutt	Lancet	Feb. 1925.
464.	H.C. Semon	Lancet	Mar. 1925.
465.	J.M.H. MacLeod	Diseases of the Skin	1933.
466.	H.A. Campbell	J. A. M. A.	Nov. 1934.
467.	C. Smart	Am. J. M. Sc.	Apr. 1865.

468./

468.	D.L.C. Bingham	Brit. M. J.	Jan. 1938.
469.	R. A. M. College	Lectures on Hygiene	1934.
470.	Field Service Regulations	Vol. II. p. 47	1935.
471.	H. Charles	J. Roy. Army M. Corps.	Feb. 1909.
472.	G. F. Mitchell	Mil Surgeon	Apr. 1908.
473.	C. B. Heald	Lancet	Feb. 1934.
474.	C. H. Melville	J. Roy. Army M. Corps.	Mar. 1911.
475.	M. Sykes	J. Roy. Army M. Corps.	Mar. 1911.
